This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

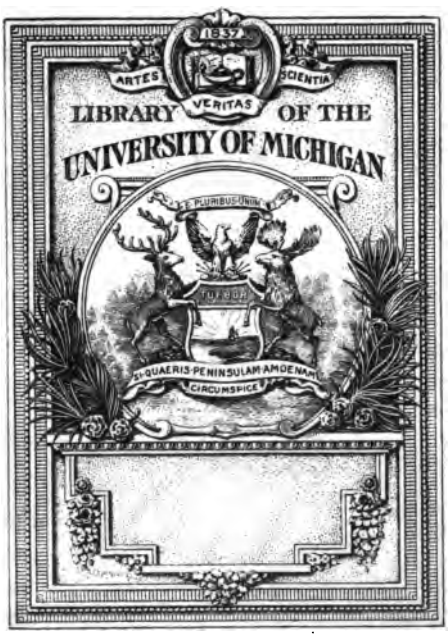
+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

A 3 9015 00382 644 6
University of Michigan - BUHR


1837
ARTES
SCIENTIA
VERITAS
LIBRARY OF THE
UNIVERSITY OF MICHIGAN
SI QUAERIS PENINSULAM AMOENAM
CIRCUMSPICE

610.5
R89
M5t

MEDICO-CHIRURGICAL TRANSACTIONS

75938

PUBLISHED BY

THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY

OF

LONDON

VOLUME THE SEVENTY-NINTH

(SECOND SERIES, VOLUME THE SIXTY-FIRST)

LONDON:
PUBLISHED FOR THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY OF LONDON BY
LONGMANS, GREEN & CO.,
PATERNOSTER ROW.

1896.

Issued from the Society's House at 20, *Hanover Square, W.*

August, 1806.

PRINTED BY ADLARD AND SON, BARTHOLOMEW CLOSE.

CONTENTS

	PAGE
List of Officers and Council	v
Referees of Papers	vi
Trustees of the Society	vi
Trustees of the Marshall Hall Memorial Fund	vi
Committee on Medical Climatology and Balneology	vii
Committee on Suspended Animation in the Drowned	vii
Library Committee	vii
House Committee	vii
Committee on Discussions	vii
List of Presidents of the Society from its Formation	viii
List of Honorary Fellows	ix
List of Foreign Honorary Fellows	x
List of Resident Fellows	xi
List of Resident Fellows arranged according to Date of Election	lvii
List of Non-resident Fellows	lxiii
Proceedings at the Annual General Meeting, March 2nd, 1896, with Report of the Council for 1895–6	lxxxiii
List of Papers	xciv
List of Plates	xcvi
Address of JONATHAN HUTCHINSON, F.R.S., President, at the Annual Meeting, March 2nd, 1896	xcvii
Papers	1
Index	337

ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF LONDON

PATRON
THE QUEEN

OFFICERS AND COUNCIL
ELECTED MARCH 2, 1896.

President
WILLIAM HOWSHIP DICKINSON, M.D.

VICE-PRESIDENTS
- JOHN HARLEY, M.D.
- JAMES EDWARD POLLOCK, M.D.
- JOHN LANGTON
- SIR WILLIAM MAC CORMAC

HON. TREASURERS
- WILLIAM SELBY CHURCH, M.D.
- J. WARRINGTON HAWARD

HON. SECRETARIES
- NORMAN MOORE, M.D.
- ROBERT WILLIAM PARKER

HON. LIBRARIANS
- SAMUEL JONES GEE, M.D.
- RICKMAN J. GODLEE, M.S.

OTHER MEMBERS OF COUNCIL
- JOHN ABERCROMBIE, M.D.
- WILLIAM EWART, M.D.
- DAVID FERRIER, M.D.
- FELIX SEMON, M.D.
- FRANCIS CHARLEWOOD TURNER, M.D.
- ARTHUR EDWARD J. BARKER
- SIR WILLIAM B. DALBY
- JOHN HAMMOND MORGAN
- EDMUND OWEN
- ROBERT ALEXANDER GIBBONS, M.D.

THE ABOVE FORM THE COUNCIL.

RESIDENT LIBRARIAN
J. Y. W. MAC ALISTER, F.S.A.

FELLOWS OF THE SOCIETY APPOINTED BY THE COUNCIL AS REFEREES OF PAPERS

FOR THE SESSION OF 1896-7.

THOMAS BARLOW, M.D.
CHARLES EDWARD BEEVOR, M.D.
STANLEY BOYD, B.S.
SIR WM. H. BROADBENT, Bart., M.D.
HENRY TRENTHAM BUTLIN
THOMAS BUZZARD, M.D.
JOHN CAVAFY, M.D.
FRANCIS HENRY CHAMPNEYS, M.D.
W. WATSON CHEYNE, C.M., F.R.S.
HENRY HUGH CLUTTON, M.B.
CHARLES J. CULLINGWORTH, M.D.
JOHN CURNOW, M.D.
CLINTON THOMAS DENT
SIR DYCE DUCKWORTH, M.D.
ALFRED LEWIS GALABIN, M.D.
FREDERICK JAMES GANT
ALFRED PEARCE GOULD, M.S.
F. DE HAVILLAND HALL, M.D.
GEORGE ERNEST HERMAN, M.B.
W. H. A. JACOBSON, M.Ch.
JEREMIAH MCCARTHY
STEPHEN MACKENZIE, M.D.
N. CHARLES MACNAMARA
EDWARD NETTLESHIP
HERBERT ISAMBARD OWEN, M.D.
JOSEPH FRANK PAYNE, M.D.
GEORGE VIVIAN POORE, M.D.
SIDNEY RINGER, M.D., F.R.S.
ARTHUR ERNEST SANSOM, M.D.
EDWARD ALBERT SCHÄFER, F.R.S.
HERBERT R. SPENCER, M.D.
AUGUSTUS WALLER, M.D., F.R.S.
WILLIAM JOHNSON WALSHAM, C.M.
WILLIAM HALE WHITE, M.D.
ALFRED WILLETT
C. THEODORE WILLIAMS, M.D.

TRUSTEES

TRUSTEES OF THE SOCIETY'S INVESTMENTS.

WALTER BUTLER CHEADLE, M.D.
FREDERICK TAYLOR, M.D.
ALFRED WILLETT

TRUSTEES FOR THE DEBENTURE-HOLDERS.

SAMUEL JONES GEE, M.D., *Chairman*
THOMAS BARLOW, M.D.
C. THEODORE WILLIAMS, M.D.

TRUSTEES OF THE MARSHALL HALL MEMORIAL FUND.

WALTER BUTLER CHEADLE, M.D.
WILLIAM OGLE, M.D.
THOMAS SMITH

COMMITTEES

COMMITTEE APPOINTED TO INVESTIGATE THE MEDICAL CLIMATOLOGY AND BALNEOLOGY OF GREAT BRITAIN AND IRELAND

WILLIAM MILLER ORD, M.D., *Chairman*

THE PRESIDENT
THE HON. SECRETARIES
ROBERT BARNES, M.D.
ROBERT LEAMON BOWLES, M.D.
JOHN MITCHELL BRUCE, M.D.
WILLIAM EWART, M.D.
W. S. LAZARUS-BARLOW, M.B.
DANIEL JOHN LEECH, M.D.
ROBERT MAGUIRE, M.D.
NORMAN MOORE, M.D.
MALCOLM ALEXANDER MORRIS
WILLIAM MURRELL, M.D.
FRANCIS G. PENROSE, M.D.
FREDERICK THOMAS ROBERTS, M.D.
FREDERICK TAYLOR, M.D.
EDMUND SYMES THOMPSON, M.D.
HOWARD HENRY TOOTH, M.D.
HERMANN WEBER, M.D.
C. THEODORE WILLIAMS, M.D.

ARCHIBALD E. GARROD, M.D., *Hon. Sec.*

COMMITTEE APPOINTED TO INVESTIGATE THE SUBJECT OF SUSPENDED ANIMATION IN THE DROWNED

EDWARD ALBERT SCHÄFER, F.R.S., *Chairman*

THE PRESIDENT
THE HON. SECRETARIES
GEORGE HARLEY, M.D., F.R.S.
HENRY POWER

THOMAS PICKERING PICK, *Hon. Sec.*

LIBRARY COMMITTEE FOR 1896–7

THE PRESIDENT
THE HON. LIBRARIANS
THE HON. SECRETARIES
WILLIAM ANDERSON.
JOHN CAVAFY, M.D.
FRANCIS H. CHAMPNEYS, M.D.
ARCHIBALD E. GARROD, M.D.
W. H. ACLAND JACOBSON, M.Ch
JOSEPH ARDERNE ORMEROD, M.D.
JOSEPH FRANK PAYNE, M.D.
GEORGE VIVIAN POORE, M.D.
D'ARCY POWER, M.B.
A. QUARRY SILCOCK, B.S.

HOUSE COMMITTEE FOR 1896–7

TIMOTHY HOLMES, *Chairman*

THE PRESIDENT
THE HON. SECRETARIES
THE SENIOR TREASURER
SIDNEY COUPLAND, M.D.
A. PEARCE GOULD, M.S.
J. WARRINGTON HAWARD
ALFRED WILLETT

ROBERT WILLIAM PARKER, *Hon. Sec.*

COMMITTEE ON DISCUSSIONS FOR 1896–7

THE PRESIDENT
THE HON. SECRETARIES
DAVID FERRIER, M.D.
JOHN H. MORGAN

FOREIGN HONORARY FELLOWS

(Limited to Twenty.)

Elected

1878 Baccelli, Guido, M.D., Rome.

1896 von Bergmann, Ernst, Berlin.

1887 Billings, John S., M.D., D.C.L.Oxon., New York.

1896 Czerny, Vincent, M.D., Heidelberg.

1883 DuBois-Reymond, Emil, M.D., Berlin.

1896 Erb, Wilhelm, M.D., Heidelberg.

1887 von Esmarch, Friedrich, M.D., Kiel.

1896 Fournier, Alfred, M.D., Paris.

1896 Koch, Robert, M.D., Berlin.

1896 Kocher, Theodore, M.D., Berne.

1868 Kölliker, Albert, Würzburg.

1896 Laveran, A., M.D., Paris.

1896 Marie, Pierre, M.D., Paris.

1896 Mitchell, Samuel Weir, M.D., Philadelphia.

1856 Virchow, Rudolph, M.D., LL.D., Berlin.

FELLOWS

OF THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY

OF LONDON

EXPLANATION OF THE ABBREVIATIONS

P.—President.	C.—Member of Council.
V.P.—Vice-President.	*Sci. Com.*—Member of a Scientific Committee.
T.—Treasurer.	*Ho. Com.*—Member of House Committee.
L.—Hon. Librarian.	*Lib. Com.*—Member of Library Committee.
S.—Hon. Secretary.	*Bldg. Com.*—Member of Building Committee.
	Dis. Com.—Member of Discussions Committee.

The abbreviations *Trans.* and *Pro.*, followed by figures, show the number of Papers which have been contributed to the *Transactions* or *Proceedings* by the Fellow whose name they follow. *Referee, Sci. Com., Lib. Com., Bldg. Com., Ho. Com.*, and *Dis. Com.*, with the dates of office, are attached to the names of those who have served as Referees of papers and on the Committees of the Society.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Those marked thus (*) have paid the Composition Fee entitling them to receive the *Transactions*.

RESIDENT FELLOWS

[N.B.—Fellows are reminded that they are, themselves, responsible for the correctness of the descriptions in the following lists, and it is particularly requested that any change of Title, Appointment, or Residence may be communicated to the Hon. Secretaries before the 1st of July in each year.]

Elected

†1877 ABERCROMBIE, JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square. C. 1896—. *Trans.* 1.

1885 ABRAHAM, PHINEAS S., M.A., M.D., Dermatologist to the West London Hospital; 2, Henrietta street, Cavendish square.

Elected

1885 ACLAND, THEODORE DYKE, M.D., Physician to St. Thomas's Hospital, and Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 74, Brook street, Grosvenor square.

†1852 ADAMS, WILLIAM, Consulting Surgeon to the Great Northern Central Hospital, the National Hospital for the Paralysed and Epileptic, and the National Orthopædic Hospital; 7, Loudoun road, St. John's Wood. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 12, Ladbroke terrace, Notting hill.

1879 ALLCHIN, WILLIAM HENRY, M.D., F.R.S. Ed., Physician to the Westminster Hospital; 5, Chandos street, Cavendish square.

1890 ALLINGHAM, HERBERT WILLIAM, Assistant Surgeon to St. George's Hospital; 25, Grosvenor street, Grosvenor square.

†1863 ALTHAUS, JULIUS, M.D., Consulting Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 48, Harley street, Cavendish square. *Trans.* 2.

1888 ANDERSON, JOHN, M.D., C.I.E., Physician to the Seamen's Hospital, Greenwich; 9, Harley street, Cavendish square.

1890 ANDERSON, WILLIAM, Surgeon to St. Thomas's Hospital; Professor of Anatomy to the Royal Academy of Arts; 2, Harley street, Cavendish square. *Lib. Com.* 1896—.

1891 ANDREWES, FREDERICK WILLIAM, M.B., 35, Welbeck street, Cavendish square.

1888 ARKLE, CHARLES, M.D., Assistant Physician to Charing Cross Hospital; 66, Wimpole street, Cavendish square.

1893 BAILEY, ROBERT COZENS, M.S., 21, Welbeck street, Cavendish square.

1891 BAKER, CHARLES ERNEST, M.B., 5, Gledhow gardens, South Kensington.

1895 BALDWIN, GERALD R., 33, Brompton square.

1887 BALL, JAMES BARRY, M.D., Physician to the West London Hospital; 12, Upper Wimpole street, Cavendish square.

Elected

1885 BALLANCE, CHARLES ALFRED, M.S., Assistant Surgeon to St. Thomas's Hospital and to the Hospital for Sick Children, Great Ormond street; Surgeon to the National Hospital for the Paralysed and Epileptic, Queen square; 106, Harley street, Cavendish square. *Trans.* 3.

1879 BARKER, ARTHUR EDWARD JAMES, Professor of the Principles and Practice of Surgery and Professor of Clinical Surgery at University College, and Surgeon to University College Hospital, London; 87, Harley street, Cavendish square. C. 1895—. *Trans.* 7.

†1876 BARLOW, THOMAS, M.D., B.S., *Trustee for Debenture-holders*; Physician-in-Ordinary to H. M.'s Household; Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond street; 10, Wimpole street, Cavendish square. C. 1892. *Referee*, 1896—. *Trans.* 2.

1893 BARRETT, HOWARD, 49, Gordon square.

1880 BARROW, A. BOYCE, Surgeon to King's College Hospital; 37, Wimpole street, Cavendish square.

1859 BARWELL, RICHARD, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 12. *Pro.* 1.

†1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Medicine in University College, London; Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic; 8A, Manchester square. C. 1885. *Referee*, 1886—96. *Trans.* 2.

1890 BATEMAN, WILLIAM A. F., Bridge House, Richmond, Surrey.

1891 BATTEN, FREDERICK E., M.D., B.C.Cantab., 124, Harley street.

1875 BEACH, FLETCHER, M.B., Winchester House, Kingston Hill [64, Welbeck street].

1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Physician to the City of London Hospital for Diseases of the Chest, and Physician to the Great Northern Central Hospital; 23, Upper Berkeley street.

Elected

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-7. *Referee*, 1873-5. *Trans.* 1.

1880 BEEVOR, CHARLES EDWARD, M.D., Physician for Out-patients to the National Hospital for the Paralysed and Epileptic, and to the Great Northern Hospital; 33, Harley street, Cavendish square. *Referee*, 1896—. *Trans.* 1

1896 BELBEN, FRANK, M.B., St. Bartholomew's Hospital.

1883 BENNETT, STORER, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to, and Lecturer on Dental Surgery and Pathology at, the Dental Hospital of London; 17, George street, Hanover square.

1877 BENNETT, WILLIAM HENRY, Surgeon to St. George's Hospital; 1, Chesterfield street, Mayfair. C. 1893-4. *Referee*, 1892-93. *Trans.* 4.

†1845 BERRY, EDWARD UNWIN, 17, Sherriff road, West Hampstead.

1885 BERRY, JAMES, B.S., Demonstrator of Anatomy, St. Bartholomew's Hospital; Surgeon to, and Lecturer on Clinical Surgery at, the Royal Free Hospital; 60, Welbeck street, Cavendish square.

1893 BIDWELL, LEONARD A., Senior Assistant Surgeon to the West London Hospital; 59, Wimpole street.

†1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

†1851 BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the Société de Chirurgie of Paris; Inspector of Anatomy for the Provinces in England and Wales; 1, Sussex gardens. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869. *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

Elected

1881 Biss, Cecil Yates, M.D., Senior Assistant Physician to, and Lecturer on Pharmacology and Therapeutics at, the Middlesex Hospital; Physician to the Hospital for Consumption, Brompton; 135, Harley street, Cavendish square. *Trans.* 2.

†1865 Blandford, George Fielding, M.D., late Lecturer on Psychological Medicine at St. George's Hospital; 48, Wimpole street, Cavendish square. C. 1883-4.

1891 Bokenham, Thomas Jessopp, 10, Devonshire street, Portland place.

1890 Bostock, R. Ashton, Surgeon, Scots Guards, 73, Onslow gardens, Brompton.

1882 Bowlby, Anthony Alfred, Assistant Surgeon to St. Bartholomew's Hospital; 24, Manchester square. *Trans.* 6.

*1870 Bowles, Robert Leamon, M.D., 16, Upper Brook street, Grosvenor square. *Sci. Com.* 1896—. *Trans.* 2.

1886 Boxall, Robert, M.D., Assistant Obstetric Physician to, and Lecturer on Practical Midwifery at, the Middlesex Hospital; 29, Weymouth street, Portland place.

1884 Boyd, Stanley, M.B., Surgeon to, and Lecturer on Anatomy at, the Charing Cross Hospital; Surgeon to the Paddington Green Children's Hospital; 134, Harley street, Cavendish square. *Referee*, 1895—. *Trans.* 1.

1890 Bradford, John Rose, M.D., D.Sc., F.R.S., Assistant Physician to University College Hospital; 52, Upper Berkeley street, Portman square.

1883 Bradshaw, James Dixon, M.B., Savile Club, Piccadilly, W.

1890 Brinton, Roland Danvers, M.D., 8, Queen's Gate terrace.

Elected

†1868 BROADBENT, SIR WILLIAM HENRY, Bart., M.D., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; Physician in Ordinary to H.R.H. the Prince of Wales; 84, Brook street, Grosvenor square. C. 1885. *Referee*, 1881-4, 1891—. *Trans.* 5.

†1851 BRODHURST, BERNARD EDWARD, Surgeon to the Royal Orthopædic Hospital and to the Royal Hospital for Incurables; Consulting Surgeon, Belgrave Hospital for Children; Corresponding Member, Société de Chirurgie, Paris, and of the Academy of Sciences, Rome; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1891 BRODIE, CHARLES GORDON, Assistant Surgeon, North-West London Hospital; 30, Harley street, Cavendish square.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 3, Chesterfield street, Mayfair. *Trans.* 1.

1880 BROWNE, JAMES WILLIAM, M.B., 160, Holland Park avenue.

1881 BROWNE, OSWALD AUCHINLECK, M.A., M.B., Physician to the Royal Hospital for Diseases of the Chest; 43, Bedford square.

1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; Physician to the Hospital for Consumption, Brompton; 23, Harley street. C. 1892. S. 1893-5. *Sci. Com.* 1889—. *Referee*, 1886-91. *Lib. Com.* 1888-91. *Trans.* 3.

1871 BRUNTON, THOMAS LAUDER, M.D., D.Sc., LL.D., F.R.S., Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888-9. *Referee*, 1880-87. *Lib. Com.* 1882-7. *Trans.* 1.

Elected

1860 BRYANT, THOMAS, Consulting Surgeon to Guy's Hospital; Member of the Société de Chirurgie, Paris; 65, Grosvenor street, Grosvenor square. C. 1873-4. V. P. 1885-6. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 15. *Pro.* 1.

1889 BULL, WILLIAM CHARLES, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 35, Clarges street, Piccadilly.

1893 BURGHARD, FRÉDÉRIC FRANÇOIS, M.D., M.S., Assistant Surgeon to King's College Hospital; 46, Weymouth street, Portland place.

1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Surgeon to the Grosvenor Hospital for Women and Children; 76, Brook street, Grosvenor square.

1873 BUTLIN, HENRY TRENTHAM, D.C.L., Surgeon to St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887-8. *Referee*, 1893—. *Trans.* 4. *Pro.* 1.

1896 BUTTAR, CHARLES F., M.B., 2, Princes square, Bayswater. *Pro.* 1.

1883 BUXTON, DUDLEY WILMOT, M.D., B.S., Administrator, and Teacher of the Use, of Anæsthetics, in University College Hospital; Anæsthetist to the National Hospital for the Paralysed and Epileptic, Queen's square, and to the London Dental Hospital; 82, Mortimer street, Cavendish square.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square. C. 1885-6. *Referee*, 1887—.

1890 CAGNEY, JAMES, M.A., M.D., in charge of Electrical Department, St. Mary's Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 93, Wimpole street, Cavendish square. *Trans.* 1.

1885 CAHILL, JOHN, 12, Seville street, Lowndes square.

Elected

1893 CALEY, HENRY ALBERT, M.D., Medical Registrar and Joint Medical Tutor, St. Mary's Hospital; 24, Upper Berkeley street, Portman square.

1887 CALVERT, JAMES, M.D., 36, Queen Anne street, Cavendish square.

1888 CARLESS, ALBERT, M.S., Assistant Surgeon to King's College Hospital; 10, Welbeck street.

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde Park.

1853 CARTER, ROBERT BRUDENELL, Consulting Ophthalmic Surgeon to St. George's Hospital; 31, Harley street, Cavendish square. *Trans.* 1.

1888 CAUTLEY, EDMUND, M.D., B.C., 15, Upper Brook street.

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. C. 1887. *Referee*, 1896—. *Lib. Com.* 1888—. *Trans.* 1.

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital, Consulting Physician to the London Fever Hospital, and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7. *Lib. Com.* 1886-7. *Trans.* 2.

1879 CHAMPNEYS, FRANCIS HENRY, M.D., Physician-Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; 42, Upper Brook street, Grosvenor square. *Referee*, 1891—. *Lib. Com.* 1885—. *Trans.* 8.

1868 CHEADLE, WALTER BUTLER, M.D., *Trustee;* Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. C. 1890-91. *Sci. Com.* 1889-95. *Bldg. Com.* 1889-92. *Referee*, 1885. *Trans.* 1.

Elected

1879 CHEYNE, WILLIAM WATSON, M.B., F.R.S., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; 75, Harley street, Cavendish square. *Referee*, 1894—. *Lib. Com.* 1886-8, 1891-6. *Trans.* 1.

1890 CHILDS, CHRISTOPHER, M.D., 10, Manchester square.

1886 CHRISTOPHERSON, JOHN BRIAN, M.B., B.C., 5, Staple Inn.

1866 CHURCH, WILLIAM SELBY, M.D., *Hon. Treasurer*, Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. V.P. 1892-4. T. 1894—. *Referee*, 1874-81.

1879 CLARK, ANDREW, Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square.

1882 CLARKE, ERNEST, M.D., B.S., Surgeon to the Central London Ophthalmic Hospital; Ophthalmic Surgeon to the Miller Hospital; 3, Chandos street, Cavendish square.

1890 CLARKE, JAMES JACKSON, M.B., Curator of the Museum and Pathologist to St. Mary's Hospital, 9, Old Cavendish street, Cavendish square.

†1848 CLARKE, JOHN, M.D., 42, Hertford street, Mayfair. C. 1866.

1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the West London Hospital; 51, Harley street, Cavendish square. *Trans.* 1.

†1879 CLUTTON, HENRY HUGH, M.A., M.B., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Surgeon to the Victoria Hospital for Children; 2, Portland place. *Referee*, 1896—. *Trans.* 1.

1888 COCK, FREDERICK WILLIAM, M.D., 1, Porchester Houses, Porchester square.

Elected

1865 COOPER, ALFRED, Consulting Surgeon to the West London Hospital; Senior Surgeon to St. Mark's Hospital; 9, Henrietta street, Cavendish square.

1889 COSENS, CHARLES HENRY, 49, Oxford terrace, Hyde Park,

1892 COTTERELL, EDWARD, Surgeon for Out-patients, London Lock Hospital; Surgeon to the Cancer Hospital; Surgeon to the West-End Hospital for Epilepsy and Diseases of the Nervous System; 5, West Halkin street, Belgrave square. *Trans.* 1.

†1860 COUPER, JOHN, Surgeon to the Royal London Ophthalmic Hospital, and Consulting Surgeon to the London Hospital; 80, Grosvenor street. C. 1876. *Referee*, 1882-3.

1877 COUPLAND, SIDNEY, M.D., Physician to, and Joint Lecturer on Practical Medicine at, the Middlesex Hospital; 16, Queen Anne street, Cavendish square. C. 1893-4. *Referee*, 1892-3. *Ho. Com.*, 1895—.

†1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Surgeon to the Victoria Hospital for Children; 19, Harley street, Cavendish square. C. 1882-3.

1878 CRICHTON-BROWNE, SIR JAMES, M.D., LL.D., F.R.S. Lord Chancellor's Visitor in Lunacy; 61, Carlisle place Mansions, Victoria street.

1874 CRIPPS, WILLIAM HARRISON, Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. C. 1890-91. *Trans.* 1.

1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; late Physician to the East London Hospital for Children; 121, Harley street, Cavendish square. *Trans.* 3.

1868 CROFT, JOHN, Consulting Surgeon to St. Thomas's Hospital; 6, Mansfield street, Cavendish square. C. 1884. V.P. 1890. *Referee*, 1885-88. *Lib. Com.* 1877-8. *Trans.* 2.

Elected

1890 CROWLE, THOMAS HENRY RICKARD, 56, Harley street, Cavendish square.

1888 CULLINGWORTH, CHARLES JAMES, M.D., Obstetric Physician and Lecturer on Midwifery at St. Thomas's Hospital; 46, Brook street, Grosvenor square. *Referee*, 1896—.

1879 CUMBERBATCH, A. ELKIN, M.B., Aural Surgeon to St. Bartholomew's Hospital, and to the National Hospital for the Paralysed and Epileptic; 80, Portland place.

1873 CURNOW, JOHN, M.D., Professor of Anatomy in King's College, London, and Physician to King's College Hospital; Senior Physician to the Seamen's Hospital; 11, Wimpole street, Cavendish square. *Referee*, 1884—.

1886 DAKIN, WILLIAM RADFORD, M.D., Obstetric Physician to, and Lecturer in Midwifery at, St. George's Hospital, and Physician to the General Lying-in Hospital; 18, Grosvenor street, Grosvenor square.

1872 DALBY, SIR WILLIAM BARTLETT, M.B., Consulting Aural Surgeon to St. George's Hospital; 18, Savile row. C. 1896—. *Trans.* 3.

1891 DALTON, NORMAN, M.D., Physician to King's College Hospital; 4, Mansfield street, Cavendish square.

1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 36, Harley street, Cavendish square. C. 1892-3. *Referee*, 1890-91. *Trans.* 3.

1889 DEAN, HENRY PERCY, M.S., Assistant Surgeon to the London Hospital; 84, Wimpole street, Cavendish square.

1878 DENT, CLINTON THOMAS, Surgeon to, and Lecturer on Practical Surgery at, St. George's Hospital; 61, Brook street. C. 1890. *Bldg. Com.* 1890-2. *Referee*, 1892—. *Trans.* 4.

1891 DE SANTI, PHILIP ROBERT WILLIAM, Assistant Surgeon and Aural Surgeon to the Westminster Hospital, 91, Harley street.

Elected

1894 DICKINSON, THOMAS VINCENT, M.D., 33, Sloane street.

†1859 DICKINSON, WILLIAM HOWSHIP, M.D., *President*; Consulting Physician to St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. P. 1896. C. 1874-5. V. P. 1887. *Referee*, 1869-73. 1882-6. *Sci. Com.* 1867, 1879, 1889-96. *Trans.* 15.

†1891 DICKINSON, WILLIAM LEE, M.D., Assistant Physician to St. George's Hospital; 9, Chesterfield street, Mayfair.

1889 DODD, HENRY WORK, Assistant Surgeon to the Royal Free Hospital, and to the Royal Westminster Ophthalmic Hospital; Ophthalmic Surgeon to the West-End Hospital for Nervous Diseases; 136, Harley street, Cavendish square.

1888 DONELAN, JAMES, M.B., M.C., Physician to the Italian Hospital, Queen square; 2, Upper Wimpole street, Cavendish square.

1879 DONKIN, HORATIO BRYAN, M.D.Oxon., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 108, Harley street, Cavendish square.

1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. C. 1893-94. *Lib. Com.* 1891-3. *Trans.* 4.

1891 DOVE, PERCY W., "Carshalton," Stapleton Hall Road, Stroud Green.

1895 DOWNES, JOSEPH LOCKHART, M.B., C.M., 271, Romford road.

1879 DREWITT, F. G. DAWTREY, M.D., Physician to the West London Hospital and to the Victoria Hospital for Children; 2, Manchester square.

1893 DRYSDALE, John H., 25, Welbeck street, Cavendish square.

Elected

†1865 DUCKWORTH, SIR DYCE, M.D., LL.D., Hon. Physician to H.R.H. the Prince of Wales; Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee*, 1885—. *Trans.* 2.

1876 DUDLEY, WILLIAM LEWIS, M.D., Senior Physician to the City Dispensary; 149, Cromwell road, South Kensington.

1871 DUKE, BENJAMIN, M.D., Windmill House, Clapham Common.

1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common.

1884 DUNCAN, WILLIAM, M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 6, Harley street, Cavendish square.

1887 DUNN, HUGH PERCY, Assistant Ophthalmic Surgeon to the West London Hospital; 54, Wimpole street, Cavendish square.

1874 DURHAM, FREDERIC, M.B., Senior Surgeon to the North-West London Hospital; late Surgical Registrar to Guy's Hospital; 82, Brook street, Grosvenor square.

1894 DURHAM, HERBERT EDWARD, M.B., 82, Brook street, Grosvenor Square. *Trans.* 1.

1868 EASTES, GEORGE, M.B.Lond., 35, Gloucester place, Hyde Park. C. 1892-3.

1888 ECCLES, ARTHUR SYMONS, M.B., C.M., 23, Hertford street, Mayfair.

1893 ECCLES, WILLIAM MCADAM, M.S., 124, Harley street.

1891 EDDOWES, ALFRED, M.D., 25, Old Burlington street.

1883 EDMUNDS, WALTER, M.C., 75, Lambeth Palace road, Albert Embankment. *Trans.* 3.

1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital, and to St. Peter's Hosspital for Stone; 55, Harley street, Cavendish square.

Elected

†1842 ERICHSEN, SIR JOHN ERIC, Bart., LL.D., F.R.S., M.Ch., Surgeon Extraordinary to H.M. the Queen; President of, and Emeritus Professor of Surgery in, University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. P. 1879-80. *Referee*, 1866-8, 1884-89. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1879 EVE, FREDERIC S., Surgeon to the London Hospital; Surgeon to the Evelina Hospital for Sick Children; 125, Harley street, Cavendish square. *Trans.* 2.

1877 EWART, WILLIAM, M.D., Physician to St. George's Hospital; 33, Curzon street, Mayfair. C. 1895—. *Sci. Com.* 1889—. *Trans.* 1.

1872 FAYRER, SIR JOSEPH, K.C.S.I., LL.D., M.D., F.R.S., Surgeon-General; Honorary Physician to H.M. the Queen, (Military) to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Physician to the Secretary of State for India in Council, and President of the Medical Board at the India Office; 16, Devonshire street, Portland place. C. 1888. *Referee*, 1881-7.

1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882—. *Trans.* 4.

1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Neuropathology in King's College, London, and Physician to King's College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Referee*, 1891-6. C. 1896—. *Dis. Com.* 1896—. *Trans.* 2.

1889 FIELD, GEORGE P., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital, and Dean of the Medical School; 34, Wimpole street, Cavendish square.

1866 FITZ-PATRICK, THOMAS, A.M., M.D., 30, Sussex gardens, Hyde Park. C. 1894-6.

1891 FLETCHER, HERBERT MORLEY, M.D., 98, Harley street, Cavendish square.

Elected

1896 FORESTIER, HENRI, M.D., Aix-les-Bains, Savoie, France.

1892 FORSBROOK, WILLIAM HENRY RUSSELL, M.D., 139, Buckingham Palace road.

1882 FOWLER, JAMES KINGSTON, M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician for Skin Diseases to the Westminster Hospital, and to the Skin Department of the Paddington Green Hospital for Children; late Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

1896 FREYER, P. J., Surg.-Lieut.-Colonel, I.M.S., M.A., M.D., 46, Harley street.

1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's Park.

1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, Mayfair.

1894 FURNIVALL, PERCY, 34, Adelaide road, South Hampstead.

†1874 GALABIN, ALFRED LEWIS, M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; 49, Wimpole st., Cavendish square. C. 1892. *Referee*, 1882-91, 1896—. *Lib. Com.* 1883-4. *Trans.* 2.

1895 GALLOWAY, JAMES, M.D., 21, Queen Anne street, Cavendish square.

1883 GALTON, JOHN CHARLES, M.A., F.L.S., 10, Upper Cheyne row, Chelsea.

1865 GANT, FREDERICK JAMES, Consulting Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. *Referee*, 1886—. *Lib. Com.* 1882-5. *Trans.* 3.

†1854 GARROD, SIR ALFRED BARING, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 9.

Elected

1886 GARROD, ARCHIBALD EDWARD, M.D., Medical Registrar and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Assistant Physician to the Hospital for Sick Children, Great Ormond street; 9, Chandos street, Cavendish square. *Sci. Com.* 1889—. *Lib. Com.* 1896—. *Trans.* 5.

1887 GAY, JOHN, 119, Upper Richmond road, Putney.

†1866 GEE, SAMUEL JONES, M.D., *Hon. Librarian; Chairman of Trustees for Debenture-holders;* Physician to St. Bartholomew's Hospital; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887—. *Sci. Com.* 1879. *Bldg. Com.* 1889-92. *Referee,* 1885-7. *Lib. Com.* 1871-6. *Trans.* 1.

1885 GELL, HENRY WILLINGHAM, M.B., 36, Hyde Park square.

1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place. C. 1896—. *Trans.* 1.

1872 GILBART-SMITH, THOMAS, M.D., Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. C. 1890. *Trans.* 1.

1893 GILES, ARTHUR EDWARD, M.D., B.Sc., 58, Harley street.

1894 GILL, RICHARD, 72, Wimpole street.

1877 GODLEE, RICKMAN JOHN, M.S., *Hon. Librarian;* Surgeon to University College Hospital, and Professor of Clinical Surgery in University College, London; Surgeon to the Hospital for Consumption, Brompton; Consulting Surgeon to the North-Eastern Hospital for Children; 19, Wimpole street, Cavendish square. S. 1892-4. L. 1895—. *Referee,* 1886-91. *Trans.* 9.

†1870 GODSON, CLEMENT, M.D., Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.

1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Surgeon to, and Lecturer on Physiology at, Guy's Hospital; 12, Queen Anne street, Cavendish square. *Trans.* 1.

1895 GOODALL, EDWARD WILBERFORCE, M.B., B.S., Eastern Hospital, Homerton.

Elected

1883 GOODHART, JAMES FREDERIC, M.D., Physician to Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 25, Portland place. *Lib. Com.* 1893—.

1889 GOODSALL, DAVID HENRY, Surgeon to the Metropolitan Hospital; Surgeon to St. Mark's Hospital; 17, Devonshire place, Upper Wimpole street.

1895 GOSSAGE, ALFRED MILNE, M.B., 54, Upper Berkeley street.

1877 GOULD, ALFRED PEARCE, M.S., Surgeon to the Middlesex Hospital; 10, Queen Anne street, Cavendish square. C. 1892-3. *Referee*, 1895—. *Ho. Com.* 1891—. *ib. Com.* 1891. *Trans.* 2.

1891 GOW, WILLIAM J., M.D., Assistant Obstetric Physician to St. Mary's Hospital; Obstetric Physician to the Royal Hospital for Women and Children; Physician to Out-Patients, Queen Charlotte's Lying-in Hospital; 27, Weymouth street, Portland place.

1873 GOWERS, WILLIAM RICHARD, M.D., F.R.S., Consulting Physician to University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. C. 1891. *Referee* 1888-90. *Lib. Com.* 1884-6. *Trans.* 7.

†1851 GOWLLAND, PETER YEAMES, late Senior Surgeon to St. Mark's Hospital; Brigade Surgeon Hon. Artillery Company; 163, Gloucester terrace, Hyde park.

1892 GRANT, J. DUNDAS, M.A., M.D., 8, Upper Wimpole street, Cavendish square.

1868 GREEN, T. HENRY, M.D., Physician to the Charing Cross Hospital, and to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5.

1885 GRIFFITH, WALTER SPENCER ANDERSON, M.D., Assistant Physician-Accoucheur, St. Bartholomew's Hospital; Physician to Queen Charlotte's Lying-in Hospital; 96, Harley street, Cavendish square.

Elected

1868 GRIGG, WILLIAM CHAPMAN, M.D., Obstetric Physician to the Out-patients at the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in Hospital; Joint Lecturer on Forensic Medicine at the Westminster Hospital Medical School; 27, Curzon street, Mayfair.

1889 GUBB, ALFRED S., M.D., 29, Gower street.

1883 GUNN, ROBERT MARCUS, M.A., M.B., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the National Hospital for the Paralysed and Epileptic; Assistant Ophthalmic Surgeon to University College Hospital; 54, Queen Anne street, Cavendish square.

1890 GUTHRIE, LEONARD GEORGE, M.D., B.Ch., Physician to the Regent's Park Hospital for Epilepsy and Paralysis; Assistant Physician to the North-West London Hospital; Assistant Physician to the Children's Hospital, Paddington Green; 15, Upper Berkeley street, Portman square.

1886 HABERSHON, SAMUEL HERBERT, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 70, Brook street, Grosvenor square.

1885 HAIG, ALEXANDER, M.D., Physician to the Metropolitan Hospital, and to the Royal Hospital for Children and Women; 7, Brook street, Grosvenor square. *Trans.* 6.

1890 HALE, CHARLES DOUGLAS BOWDICH, M.D., 3, Sussex place, Hyde Park.

1881 HALL, FRANCIS DE HAVILLAND, M.D., Physician to the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square. *Referee*, 1893—.

1891 HAMER, WILLIAM HEATON, M.D., 73, Dartmouth Park Hill, Highgate.

1889 HANDFIELD-JONES, MONTAGU, M.D., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Mary's Hospital; Physician to the British Lying-in Hospital; 35, Cavendish square.

Elected

†1856 HARE, CHARLES JOHN, M.D., Consulting Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4. T. 1887-94. V.P. 1894-5. *Bldg. Com.* 1889-92.

†1857 HARLEY, GEORGE, M.D., F.R.S., 25, Harley street, Cavendish square. C. 1871-2. V.P. 1891-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3, 1866, 1889—. *Trans.* 1.

†1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. V.P. 1895—. *Referee*, 1871-4, 1882-95. *Sci. Com.* 1879. *Trans.* 10.

1893 HARLEY, VAUGHAN, M.D., 25, Harley street, Cavendish square.

1892 HAROLD, JOHN, 91, Harley street, Cavendish square.

1880 HARRIS, VINCENT DORMER, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 31, Wimpole street, Cavendish square.

1870 HARRISON, REGINALD, 6, Lower Berkeley Street, Portman square. C. 1894-5. *Trans.* 2.

1870 HAWARD, J. WARRINGTON, *Hon. Treasurer;* Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 16, Savile row, Burlington Gardens. C. 1885. S. 1888-91. V.P. 1894-5. T. (June) 1895—. *Lib. Com.* 1881-4. *Sci. Com.* 1889-91. *Bldg. Com.* (*Sec.*) 1889-92. *Ho. Com.* 1892—. *Trans.* 2.

1891 HAWKINS, HERBERT PENNELL, M.D., B.C., Assistant Physician to St. Thomas's Hospital; 109, Harley street, Cavendish square.

1875 HAYES, THOMAS CRAWFORD, M.A., M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital, and Lecturer on Practical Obstetrics in King's College; Physician for Diseases of Women to the Royal Free Hospital; 17, Clarges street, Piccadilly.

Elected

1860 HAYWARD, HENRY HOWARD, Consulting Surgeon Dentist to St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

1891 HAYWARD, JOHN ARTHUR, M.D., 58, Brook street. *Pro.* 1.

†1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. V.P. 1889. *Lib. Com.* 1870-8. *Trans.* 3.

1895 HENDERSON, EDWARD ERSKINE, B.A., M.B., B.C., Bruntsfield Lodge, Homefield road, Bromley, Kent.

1882 HENSLEY, PHILIP JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Referee*, 1892—. *Trans.* 1.

1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.

1891 HERRING, HERBERT T., M.B., B.S., 50, Harley street, Cavendish square.

1883 HERRINGHAM, WILMOT PARKER, M.D., Medical Registrar, St. Bartholomew's Hospital; 13, Upper Wimpole street, Cavendish square. *Trans.* 1.

1893 HERSCHELL, GEORGE, M.D., 25, Queen Anne street, Cavendish square.

1887 HEWITT, FREDERIC WILLIAM, M.D., Anæsthetist to, and Instructor in Anæsthetics at, the London Hospital; Chloroformist to, and Lecturer on Anæsthetics at, Charing Cross Hospital; Anæsthetist at the Dental Hospital of London; 10, George street, Hanover square. *Trans.* 2.

Elected

1873 HIGGENS, CHARLES, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. C. 1894-5. *Trans.* 2.

1890 HILL, G. WILLIAM, M.D., B.Sc., 24, Wimpole street, Cavendish square.

†1856 HOLMES, TIMOTHY, M.A., Consulting Surgeon to St. George's Hospital; Corresponding Member of the Société de Chirurgie, Paris; 6, Sussex place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. P. 1890-92. *Bldg. Com.* (*Chairman*) 1889-92. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5, 1892-5. *Ho. Com.* 1892—. *Trans.* 8.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., Senior Physician to the West London Hospital; 43, Green street, Park lane.

1883 HORSLEY, VICTOR ALEXANDER HADEN, F.R.S., Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; Professor of Pathology in University College, London; 25, Cavendish square. *Trans.* 1.

1896 HORTON-SMITH, PERCIVAL, M.B., 15, Upper Brook street.

1892 HOWARD, R. J. BLISS, M.D., 31, Queen Anne street, Cavendish square.

1874 HOWSE, HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. C. 1890. *Sci. Com.* 1879. *Referee*, 1887-89. *Trans.* 3.

1886 HUDSON, CHARLES ELLIOTT LEOPOLD BARTON, Assistant Surgeon and Surgeon to Aural Department, Middlesex Hospital; Surgeon to Aural Department to the Hospital for Sick Children, Great Ormond Street; 16, Harley street, Cavendish square.

Elected

1889 HUNTER, WILLIAM, M.D., Senior Assistant Physician to the London Fever Hospital; 54, Harley street.

1873 HUNTER, SIR W. GUYER, M.D., K.C.M.G., Hon. Surgeon to H.M. the Queen; formerly Principal of, and Professor of Medicine in, Grant Medical College, and Vice-Chancellor of the University, Bombay; Surgeon-General (Retired) Bombay Army; Consulting Physician to Charing Cross Hospital; 21, Norfolk crescent, Hyde Park.

†1856 HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Senior Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. P. 1894-5. *Referee*, 1876-81, 1883-94. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.

1888 HUTCHINSON, JONATHAN, Jun., Assistant Surgeon to the London Hospital; 1, Park crescent. *Trans.* 1.

1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Consulting Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square. C. 1889.

†1841 JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.

1883 JACOBSON, WALTER HAMILTON ACLAND, M.A., M.B., M.Ch., Assistant Surgeon and Lecturer on Anatomy to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 66, Great Cumberland place, Hyde Park. *Referee*, 1895—. *Lib. Com.* 1896—. *Trans.* 2.

1892 JAMES, EDWIN MATTHEWS, Belgrave Mansions, Grosvenor gardens.

1884 JESSETT, FREDERIC BOWREMAN, Surgeon to the Cancer Hospital, Brompton; 1, Buckingham Palace Mansions.

1883 JESSOP, WALTER H. H., M.B., Ophthalmic Surgeon to St. Bartholomew's Hospital; 73, Harley street.

Elected

1881 JOHNSON, GEORGE LINDSAY, M.D., Cortina, Netherhall gardens, South Hampstead, and 14, Stratford place, Oxford street.

1889 JOHNSON, RAYMOND, M.B., B.S., Assistant Surgeon to University College Hospital; Surgeon to the Victoria Hospital for Children; 20, Weymouth street. *Trans.* 1.

1884 JOHNSTON, JAMES, M.D., 53, Prince's square, Bayswater.

1887 JONES, HENRY LEWIS, M.D., Medical Officer in charge of Electrical Department at St. Bartholomew's Hospital; 9, Upper Wimpole street, Cavendish square.

1881 JULER, HENRY EDWARD, Ophthalmic Surgeon to St. Mary's Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Ophthalmic Surgeon to the London Lock Hospital; 23, Cavendish square.

1893 KANTHACK, ALFRED A., M.D., Lecturer on Pathology, St. Bartholomew's Hospital.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square.

1884 KESER, JEAN SAMUEL, M.D., Physician to the French Hospital; 11, Harley street, Cavendish square.

†1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens. C. 1890-91.

1881 KIDD, PERCY, M.D., Physician to the Hospital for Consumption, Brompton; Assistant Physician to the London Hospital; 60, Brook street, Grosvenor square. *Trans.* 4.

†1851 KINGDON, JOHN ABERNETHY, Consulting Surgeon to the Bank of England; 2, Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1895 LANE, JAMES ERNEST, 46, Queen Anne street, Cavendish square.

1884 LANE, WILLIAM ARBUTHNOT, M.S., Lecturer on Anatomy at Guy's Hospital; Assistant Surgeon to the Hospital for Sick Children; 21, Cavendish square. *Trans.* 4.

Elected

1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 22, Cavendish square.

†1863 LANGDON-DOWN, JOHN LANGDON HAYDON, M.D., Consulting Physician to the London Hospital; 81, Harley street, Cavendish square. C. 1880. V.P. 1890-91. *Trans.* 2.

1894 LANGDON-DOWN, REGINALD LANGDON, M.B., B.C., 81, Harley street.

†1865 LANGTON, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881-2. V.P. 1895—. *Referee*, 1885-95. *Lib. Com.* 1879-80, 1888-95. *Trans.* 2.

1890 LAW, EDWARD, M.D., C.M., 35, Harley street, Cavendish square.

1888 LAWRENCE, LAURIE ASHER, 125, Harley street, Cavendish square.

1893 LAWSON, ARNOLD, 12, Harley street.

1884 LAWSON, GEORGE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Surgeon to the Royal London Ophthalmic Hospital; Surgeon to the Middlesex Hospital; 12, Harley street, Cavendish square.

1892 LEADAM, WILLIAM WARD, M.D., 80, Gloucester terrace, Hyde Park.

†1843 LEE, HENRY, Consulting Surgeon to St. George's Hospital; 61, Queenborough terrace, Hyde Park. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 14. *Pro.* 2.

1896 LEE, WILLIAM EDWARD, M.B., St. Bartholomew's Hospital.

1895 LEES, DAVID BRIDGE, M.D., 22, Weymouth street, Portland place.

Elected

1895 Leslie, Robert Murray, M.B., 58, Harley street.

1886 Lewers, Arthur Hamilton Nicholson, M.D., Obstetric Physician to the London Hospital; 72, Harley street, Cavendish square. *Trans.* 1.

1896 Lewis, Frederick Henry, M.B., St. Bartholomew's Hospital.

1878 Lister, Sir Joseph, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery in King's College, London; and Consulting Surgeon to King's College Hospital; 12, Park crescent, Regent's Park. C. 1892.

1891 Little, Ernest Muirhead, 40, Seymour street, Portman square.

1889 Little, John Fletcher, M.B., 32, Harley street, Cavendish square.

1881 Lockwood, Charles Barrett, Surgeon to the Great Northern Central Hospital; Assistant Surgeon to, and Demonstrator of Operative Surgery at, St. Bartholomew's Hospital; 19, Upper Berkeley street, Portman square. *Trans.* 4.

1881 Lucas, Richard Clement, B.S., M.B., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; Corresponding Member of the Société de Chirurgie of Paris; 50, Wimpole street, Cavendish square. *Trans.* 2.

1888 Luff, Arthur Pearson, M.D., B.Sc., Physician to Out-patients and Lecturer on Medical Jurisprudence at St. Mary's Hospital; Official Analyst to the Home Office; 31, Weymouth street, Portland place.

1887 Lush, Percy J. F., M.B., 4, Maresfield gardens, Hampstead.

†1873 MacCarthy, Jeremiah, M.A., Surgeon to the London Hospital, late Lecturer on Surgery at the London Hospital Medical College; 1, Cambridge place, Victoria road, Kensington. C. 1886-7. *Lib. Com.* 1882-5. *Referee*, 1890—.

Elected

1867 Mac Cormac, Sir William, M.A., M.Ch., D.Sc., Consulting Surgeon to St. Thomas's Hospital; 13, Harley street. C. 1884-5. V.P. 1896. *Referee*, 1889-96. *Lib. Com.* 1895. *Trans.* 1.

1894 Macfadyen, Allan, M.D., B.S., 101, Great Russell street.

1880 McHardy, Malcolm Macdonald, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Surgeon to the Royal Eye Hospital, Southwark; 5, Savile row.

1873 MacKellar, Alexander Oberlin, M.Ch., Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 79, Wimpole street, Cavendish square.

1881 Mackenzie, Stephen, M.D., Physician to the London Hospital, and Lecturer on the Principles and Practice of Medicine at the London Hospital Medical College; Physician to the Royal London Ophthalmic Hospital; 18, Cavendish square. *Referee*, 1890—. *Trans.* 1.

1879 Maclagan, Thomas John, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.

1889 MacLehose, Norman MacMillan, M.B., C.M., 13, Queen Anne street, Cavendish square.

1893 McLeod, Kenneth, M.D., 39, Clanricarde gardens, Bayswater. *Trans.* 1.

1876 Macnamara, N. Charles, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Consulting Surgeon to the Royal Westminster Ophthalmic Hospital; 13, Grosvenor street. C. 1891-2. *Referee*, 1884-90, 1895—. *Lib. Com.* 1886-90.

1881 Macready, Jonathan Forster Christian Horace, Surgeon to the Great Northern Hospital; 51, Queen Anne street, Cavendish square.

1880 Maddick, Edmund Distin, 2, Chandos street, Cavendish square.

Elected

1886 MAGUIRE, ROBERT, M.D., Physician to Out-patients and Joint Lecturer on Pathology at St. Mary's Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 4, Seymour street, Portman square. *Sci. Com.* 1889—.

1880 MAKINS, GEORGE HENRY, Assistant Surgeon to St. Thomas's Hospital; Surgeon to the Evelina Hospital for Children; 47, Charles street, Berkeley square. *Trans.* 1.

1885 MALCOLM, JOHN DAVID, M.B., C.M., Surgeon to the Samaritan Free Hospital; 13, Portman street, Portman square. *Trans.* 2.

1890 MANSON, PATRICK, M.D., C.M., LL.D., Physician to the Seamen's Hospital, Greenwich; 21, Queen Anne street, Cavendish square.

1855 MARCET, WILLIAM, M.D., F.R.S., Flowermead, Wimbledon Park. C. 1871. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

1867 MARSH, F. HOWARD, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 30, Bruton street, Berkeley square. C. 1882-3, 1889. S. 1885-7. V.P. 1891-3. *Lib. Com.* 1880-1. *Trans.* 4.

1891 MARTIN, HENRY CHARRINGTON, M.D., 27, Oxford square.

1884 MARTIN, SIDNEY HARRIS COX, M.D., F.R.S., Assistant Physician to University College Hospital, and to the Hospital for Consumption, Brompton; 10, Mansfield street, Portland place.

1892 MASTERS, JOHN ALFRED, M.D., 57, Lexham gardens, Kensington.

1891 MAY, WILLIAM PAGE, M.D., B.Sc., 38, Weymouth street.

1891 MERCIER, CHARLES ARTHUR, M.B., Lecturer on Neurology and Insanity to Westminster Hospital; 8, New Court, Lincoln's Inn, and Flower House, Southend, Catford.

Elected

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 21, Manchester square. *Trans.* 1.

1894 MICHELS, ERNST, M.D., 6, West street, Finsbury circus. *Trans.* 2.

1893 MILEY, MILES, M.B., 21, Belsize avenue, Hampstead.

1891 MOLINE, PAUL, M.B., 42, Walton street, Chelsea.

1873 MOORE, NORMAN, M.D., *Hon. Secretary,* Assistant Physician and Lecturer on Medicine to St. Bartholomew's Hospital; 94, Gloucester place, Portman square. C. 1891-2. S. 1896—. *Referee,* 1886-90. *Sci. Com.* 1889—.

1878 MORGAN, JOHN HAMMOND, M.A., Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. C. 1895—. *Dis. Com.* 1896. *Trans.* 2.

1894 MORISON, ALEXANDER, M.D., 14, Upper Berkeley street, W.

1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 8, Cavendish square. C. 1888-9. *Referee,* 1882-7. *Lib. Com.* 1895. *Trans.* 10.

1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital; 8, Harley street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.

1885 MOTT, FREDERICK WALKER, M.D., Assistant Physician to Charing Cross Hospital; Pathologist to the London County Council; 84, Wimpole street, Cavendish square.

1896 MURPHY, JAMES KEOGH, M.B., St. Bartholomew's Hospital.

†1888 MURRAY, HUBERT MONTAGUE, M.D., Physician to Outpatients, and Lecturer on Pathology at, the Charing Cross Hospital; 27, Savile row.

Elected

1880 MURRELL, WILLIAM, M.D., Physician to Out-patients, and Lecturer on Materia Medica and Therapeutics, at the Westminster Hospital; 17, Welbeck street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.

1892 MYDDELTON-GAVEY, E. HERBERT, 94, Wimpole street, Cavendish square.

1863 MYERS, ARTHUR BOWEN RICHARDS, late Brigade-Surgeon, Brigade of Guards; 43, Gloucester street, Warwick square. C. 1878-9. *Lib. Com.* 1877.

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole street, Cavendish square. *Referee*, 1892—.

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1880 OGILVIE, GEORGE, M.B., B.Sc., Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 22, Welbeck street, Cavendish square. *Trans.* 1.

1880 OGILVIE, LESLIE, M.B., B.Sc., Physician to the Paddington Green Children's Hospital; 46, Welbeck street, Cavendish square.

1891 OGLE, CYRIL, M.A., M.B., 30, Cavendish square.

1858 OGLE, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 96, Gloucester place, Portman square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.

1860 OGLE, WILLIAM, M.D., late Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.

1895 OLIVER, GEORGE, M.D., 77, Wimpole street, Cavendish square.

1892 OPENSHAW, T. HORROCKS, M.B., M.S., Assistant Surgeon to, and Lecturer on Anatomy at, the London Hospital; 16, Wimpole street, Cavendish square.

Elected

1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook street, Grosvenor square. C. 1889–90. *Sci. Com.* 1889—. *Referee*, 1884-8. *Trans.* 6.

1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Physician to St. Bartholomew's Hospital; Physician to the National Hospital for the Paralysed and Epileptic, Queen square; 25, Upper Wimpole street. *Lib. Com.* 1896. *Trans.* 1.

1875 OSBORN, SAMUEL C., 10, Maddox street, Regent street, and Maisonnette, Datchet, Berks.

1879 OWEN, EDMUND, M.B., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; Senior Surgeon to the Hospital for Sick Children, Great Ormond street; 64, Great Cumberland place, Hyde park. C. 1896—. *Trans.* 3.

1882 OWEN, HERBERT ISAMBARD, M.D., Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 40, Curzon street, Mayfair. *Bldg. Com.* 1889-92. *Referee*, 1893, 1895—.

1892 PAGE, HARRY MARMADUKE, 82, Ashley gardens, Victoria street.

1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. C. 1890-91. *Referee*, 1884-89. *Lib. Com.* 1886-8. *Trans.* 4.

†1840 PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Foreign Associate of the Académie de Médecine, Paris; 5, Park square West, Regent's park. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

Elected

1886 PAGET, STEPHEN, Surgeon to, and Surgeon to the Aural Department at, the West London Hospital; 57, Wimpole street, Cavendish square.

1895 PARKER, CHARLES ARTHUR, 41, Queen Anne street, Cavendish square.

1873 PARKER, ROBERT WILLIAM, *Hon. Secretary;* Senior Surgeon to the East London Hospital for Children; Senior Surgeon to the German Hospital; 13, Welbeck street, Cavendish square. C. 1888-9. S. 1895—. *Bldg. Com.* 1889-92. *Referee*, 1891-5. *Lib. Com.* 1885-87, 1892-5. *Ho. Com.* 1892-5. *Trans.* 4.

1889 PARSONS, J. INGLIS, M.D., Physician to Out-patients, Chelsea Hospital for Women; 3, Queen street, Mayfair.

1883 PASTEUR, WILLIAM, M.D., Assistant Physician to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 4, Chandos street, Cavendish square.

1891 PATERSON, WILLIAM BROMFIELD, 64, Brook street, Grosvenor square.

1891 PATON, EDWARD PERCY, M.D., 84, Park street, Grosvenor square.

†1865 PAVY, FREDERICK WILLIAM, M.D., LL.D., F.R.S., Consulting Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. V.P. 1893-4. *Referee*, 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Referee*, 1890—. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889—.

1894 PEGLER, L. HEMINGTON, M.D., 25, Old Burlington street.

1887 PENROSE, FRANCIS GEORGE, M.D., Assistant Physician to St. George's Hospital; 4, Harley street, Cavendish square. *Sci. Com.* 1889—.

1890 PERRY, EDWIN COOPER, M.D., Assistant Physician to, and Demonstrator of Pathology at, Guy's Hospital; The College, Guy's Hospital.

Elected

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., LL.D., 10, Henrietta street, Cavendish square.

1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Palace Court, Notting hill gate.

1888 PHILLIPS, JOHN, M.D., Assistant Obstetric Physician, King's College Hospital; Physician to the British Lying-in Hospital; 71, Grosvenor street, Grosvenor square. *Trans.* 1.

1889 PHILLIPS, SIDNEY, M.D., Senior Physician to Out-patients and Lecturer on Materia Medica at St. Mary's Hospital; Senior Physician to the London Fever Hospital, and to the Lock Hospital; 62, Upper Berkeley street, Portman square.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. V.P. 1893-4. *Referee*, 1882-3, 1891-93. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1879-81.

1884 PITT, GEORGE NEWTON, M.D., Assistant Physician to, and Pathologist at, Guy's Hospital; 24, St. Thomas's street, Southwark. *Trans.* 1.

1889 PITTS, BERNARD, M.B., M.C., Surgeon to St. Thomas's Hospital; 109, Harley street, Cavendish square.

1885 POLAND, JOHN, Surgeon to the Miller Hospital, Greenwich; 4, St. Thomas's street, Southwark.

1884 POLLARD, BILTON, B.S., Surgeon to University College Hospital, Surgeon to the North-Eastern Hospital for Children; 24, Harley street, Cavendish square. *Trans.* 1.

†1845 POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 35, Chester square. C. 1856-7. L. 1859-62. V.P. 1870-1. P. 1886-7. *Referee*, 1858, 1864-9, 1877-85. *Trans.* 5.

Elected

1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. V.P. 1896. *Referee*, 1872-81.

1894 POLLOCK, WILLIAM RIVERS, M.B., B.C., 56, Park street, Grosvenor square.

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street, Cavendish square. C. 1890-91. *Referee* 1887-89, 1892—. *Lib. Com.* 1895—. *Trans.* 2.

1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.

1867 POWELL, RICHARD DOUGLAS, M.D., Physician Extraordinary to H.M. the Queen; Physician to the Middlesex Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 62, Wimpole street, Cavendish square. S. (Oct.) 1883-5. C. 1887-8. *Referee*, 1879-83, 1886. *Trans.* 3.

1887 POWER, D'ARCY, M.A., M.B., Demonstrator of Surgery at St. Bartholomew's Hospital; Surgeon to the Victoria Hospital for Children; 10A, Chandos street, Cavendish Square. *Lib. Com.* 1896—. *Trans.* 2.

1867 POWER, HENRY, Consulting Ophthalmic Surgeon to St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde Park. C. 1882-3. V.P. 1892-3. *Referee*, 1870-81, 1891—. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1872-8.

†1857 PRIESTLEY, SIR WILLIAM OVEREND, M.D., LL.D., M.P., Consulting Physician to King's College Hospital, and to the West London Hospital and the British Lying-in Hospital; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863

Elected

1883 PRINGLE, JOHN JAMES, M.B., C.M., Lecturer on Practical Medicine, Physician in Charge of Skin Department at, the Middlesex Hospital; 23, Lower Seymour street, Portman square. *Trans.* 2.

1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital ; 20, Stratford place, Oxford street. *Trans.* 2.

1877 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 48, Brook street, Grosvenor square. C. 1893-94. *Lib. Com.* 1887-93. *Trans.* 1.

†1850 QUAIN, SIR RICHARD, Bart., M.D., (Hon.) M.D.Dublin, LL.D.Ed., F.R.S., Physician Extraordinary to H.M. the Queen; President of the General Medical Council; Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

1892 RAYNER, HENRY, M.D., 2, Harley street, Cavendish square.

1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.

1891 REECE, RICHARD JAMES, 31, Holland Villas road.

1891 REMFRY, LEONARD, M.D., Obstetrical Physician to the Great Northern Central Hospital; Assistant Obstetric Physician to, and Assistant Lecturer on Obstetric Medicine at, St. George's Hospital; 60, Great Cumberland place.

1891 RENDEL, ARTHUR BOWEN, M.A., M.B., B.C., 43, Albion street, Hyde Park.

1887 RICHARDSON, GILBERT, M.A., M.D., Hawthorn House, Putney.

1863 RINGER, SYDNEY, M.D., F.R.S., Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80, 1889—. *Trans.* 6.

Elected

1871 RIVINGTON, WALTER, M.S., Consulting Surgeon to the London Hospital; 95, Wimpole street, Cavendish square. C. 1885-6. *Trans.* 5.

1896 ROBERTS, CHARLES HUBERT, M.D., 21, Welbeck street.

1893 ROBERTS, D. WATKIN, M.D., 56, Manchester street, Manchester square.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics, and of Clinical Medicine, in University College, London; Physician to University College Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square. C. 1894-5. *Sci. Com.* 1889—.

1889 ROBERTS, SIR WILLIAM, M.D., B.A., F.R.S., 8, Manchester square. *Trans.* 2.

1890 ROLLESTON, HUMPHRY DAVY, M.D., Assistant Physician to, and Lecturer on Pathology at, St. George's Hospital; 112, Harley street, Cavendish square.

†1857 ROSE, HENRY COOPER, M.D., Consulting Surgeon to the Hampstead Dispensary; 53, Roslyn hill, Hampstead. C. 1886-7. *Trans.* 1.

1883 ROSE, WILLIAM, M.B., Professor of Clinical Surgery in King's College; Surgeon to King's College Hospital; and Consulting Surgeon to the Royal Free Hospital; 17, Harley street, Cavendish square.

1888 ROUGHTON, EDMUND WILKINSON, B.S., M.D., Warden of the College of St. Mary's Hospital; Assistant Surgeon to the Royal Free Hospital; 33, Westbourne terrace, Hyde Park. *Trans.* 1.

1882 ROUTH, AMAND JULES MCCONNEL, M.D., B.S., Obstetric Physician to Out-patients, and Lecturer on Practical Midwifery, at the Charing Cross Hospital; Physician to the Samaritan Free Hospital for Women and Children; 14A, Manchester square.

†1849 ROUTH, CHARLES HENRY FELIX, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

Elected

1891 RUSSELL, J. S. RISIEN, M.B., C.M., Assistant Physician to the Metropolitan Hospital; 4, Queen Anne street, Cavendish square.

1886 SAINSBURY, HARRINGTON, M.D., Physician to the Royal Free Hospital; Assistant Physician to the City of London Hospital for Diseases of the Chest; 63, Welbeck street, Cavendish square. *Trans.* 1.

†1869 SANSOM, ARTHUR ERNEST, M.D., Physician to the London Hospital; Consulting Physician, North-Eastern Hospital for Children; 84, Harley street, Cavendish square. C. 1887-8. *Referee*, 1889—. *Trans.* 3.

†1845 SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales; Fairlawn, Wimbledon Common. C. 1872-3.

1879 SAVAGE, GEORGE HENRY, M.D., Lecturer on Mental Diseases at Guy's Hospital; 3, Henrietta street, Cavendish square.

1883 SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology, University College, London; University College, Gower street. *Referee*, 1888—. *Sci. Com.* 1889—.

1892 SCHORSTEIN, GUSTAVE, M.A., M.B., B.Ch., D.P.H., Assistant Physician to the London Hospital, and to the Hospital for Consumption, Brompton; 11, Portland place.

1887 SCOTT, HARRY, M.D., 27, St. Ermin's mansions, Westminster.

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

†1863 SEDGWICK, WILLIAM, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.

1892 DE SEGUNDO, CHARLES SEMPILL, 2, Aldridge road villas, Westbourne park.

1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood road, Cromwell road.

Elected

1877 SEMON, FELIX, M.D., Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole street, Cavendish square. C. 1895—. *Lib. Com.* 1894. *Trans.* 3.

1894 SEWILL, JOSEPH SEFTON, 9A, Cavendish square.

1882 SHARKEY, SEYMOUR JOHN, M.D., Physician to, and Joint Lecturer on Medicine at, St. Thomas's Hospital; 22, Harley street, Cavendish square. *Trans.* 2.

1886 SHAW, LAURISTON ELGIE, M.D., Assistant Physician to Guy's Hospital; 10, St. Thomas's street, Southwark.

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.C., Assistant Surgeon to St. George's Hospital; 4, Cavendish place. *Trans.* 4.

1896 SHORE, THOMAS WILLIAM, M.D., The Warden's House, St. Bartholomew's Hospital.

1893 SIBLEY, WALTER KNOWSLEY, M.D., B.C., Senior Physician to Out-patients, North-West London Hospital; 7, Upper Brook street.

†1848 SIEVEKING, SIR EDWARD HENRY, M.D., LL.D., F.S.A. Physician-in-Ordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's and the Lock Hospitals; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. P. 1888-9. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

1886 SILCOCK, ARTHUR QUARRY, B.S., Surgeon in charge of Out-patients, St. Mary's Hospital; Surgeon to the Royal London Ophthalmic Hospital; 52, Harley street, Cavendish square. *Lib. Com.* 1895—.

†1842 SIMON, SIR JOHN, K.C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee*, 1851-3, 1866-81. *Trans.* 1.

1892 SIMS, FRANCIS MANLEY BOLDERO, 12, Hertford street, Mayfair.

1893 SISLEY, RICHARD, M.D., 11, York street, Portman square.

Elected

1894 SLATER, CHARLES, M.B., 16, Northwick terrace, St. John's Wood.

1896 SLOANE, JOHN STRETTON, M.B., 3, Montague mansions, Portman square.

1890 SMALE, MORTON, 22A, Cavendish square.

1879 SMITH, E. NOBLE, Surgeon to All Saints' Children's Hospital; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 15, Queen Anne street, Cavendish square.

1891 SMITH, G. COCKBURN, M.D., 5, Inverness gardens, Kensington.

1838 SMITH, HENRY SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

1866 SMITH, HEYWOOD, M.D., 18, Harley street, Cavendish square.

1889 SMITH, ROBERT PERCY, M.D., B.S., Resident Physician and Medical Superintendent, Bethlem Royal Hospital, St. George's road, Southwark.

1892 SMITH, SOLOMON CHARLES, M.D., 1, Montague Mansions, Portman square.

†1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.

1894 SMITH, THOMAS RUDOLPH, M.B., B.C., 5, Stratford place, Oxford street.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital Society, Greenwich.

Elected

*1874 SMITH, WILLIAM ROBERT, M.D., D.Sc., Barrister-at-Law, Professor of Forensic Medicine in, and Director of the Laboratories of State Medicine at, King's College, London; Medical Officer to the School Board for London; 74, Great Russell Street. *Trans.* 1.

1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.

1889 SPENCER, HERBERT R., M.D., B.S., Professor of Midwifery in University College; Obstetric Physician to University College Hospital; 10, Mansfield street, Portland place. *Referee*, 1894—.

1887 SPENCER, WALTER GEORGE, M.B., M.S., Assistant Surgeon to the Westminster Hospital; 35, Brook street, Grosvenor square. *Trans.* 2.

1888 SPICER, ROBERT HENRY SCANES, M.D., Physician to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck street, Cavendish square.

1890 SPICER, WILLIAM THOMAS HOLMES, M.B., 47, Welbeck street, Cavendish square.

1875 SPITTA, EDMUND JOHNSON, Ivy House, Clapham Common, Surrey.

†1851 SPITTA, ROBERT JOHN, M.D., 2, The Sweep, East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1885 SQUIRE, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption; 122, Harley street, Cavendish square. *Trans.* 2.

1896 STEPHENS, JOHN WILLIAM WATSON, M.B., B.C., 25, East Paul's Wharf.

†1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

1884 STONHAM, CHARLES, Assistant Surgeon to the Westminster Hospital, and Curator of Anatomical Museum; 4, Harley street, Cavendish square.

1896 SUTHERLAND, GEORGE ALEXANDER, M.D., 9, Old Cavendish street.

Elected

†1871 SUTHERLAND, HENRY, M.D., Physician to Newland's House and Otto House Private Asylums; 6, Richmond terrace, Whitehall.

1883 SUTTON, JOHN BLAND, Assistant Surgeon to the Middlesex Hospital; 48, Queen Anne street, Cavendish square. *Trans.* 6.

1896 SWAN, CHARLES ROBERT JOHN ATKIN, M.B., B.Ch., 4, Devonport street, Hyde Park.

1890 SYERS, HENRY WALTER, M.D., 4c, Oxford and Cambridge Mansions, Chapel street.

1886 SYMONDS, CHARTERS JAMES, M.S., Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; 26, Weymouth street, Portland place.

1875 TAY, WARREN, Senior Surgeon to the London Hospital; Surgeon to the Royal London Ophthalmic Hospital; Consulting Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Diseases of the Skin, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., *Trustee;* Physician to, and Lecturer on Medicine at, Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 20, Wimpole street, Cavendish square. S. 1889-93. C. 1894-5. *Sci. Com.* 1889—. *Referee,* 1887-8. *Trans.* 3.

1893 TAYLOR, JAMES, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 49, Welbeck street, Cavendish square. *Trans.* 1.

1890 TAYLOR, SEYMOUR, M.D., Assistant Physician, West London Hospital; 16, Seymour street, Portman square.

1859 TEGART, EDWARD, 60, Scarsdale Villas, Kensington. C. 1888-9.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. C. 1893-4. *Trans.* 13.

†1862 THOMPSON, EDMUND SYMES, M.D., Consulting Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Sci. Com.* 1889—. *Referee,* 1876-7. *Trans.* 1.

Elected

†1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Member of the Société de Chirurgie, Paris; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 8.

†1862 THOMPSON, REGINALD EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1892 THOMSON, ST. CLAIR, M.D., 28, Queen Anne street, Cavendish square. *Trans.* 1.

1892 THORNE, WILLIAM BEZLY, M.D., 53, Upper Brook Street.

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Consulting Surgeon to the Grosvenor Hospital for Women, and to the New Hospital for Women; 49, Montagu square. C. 1891. *Lib. Com.* 1886-90, 1893-95. *Trans.* 5.

1889 TIRARD, NESTOR ISIDORE CHARLES, M.D., Professor of Materia Medica and Therapeutics, King's College; Physician to King's College Hospital, and Physician to the Evelina Hospital for Sick Children; 74, Harley street, Cavendish square.

1872 TOMES, CHARLES SISSMORE, M.A., F.R.S., 37, Cavendish square. C. 1887. *Lib. Com.* 1879.

1882 TOOTH, HOWARD HENRY, M.D., Assistant Physician, late Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Physician to the Metropolitan Hospital; Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen square; 34, Harley street, Cavendish square. *Sci. Com.* 1896—.

1879 TREVES, FREDERICK, Surgeon to, and Lecturer on Surgery at, the London Hospital; 6, Wimpole street, Cavendish square. C. 1895. *Referee*, 1890—. *Sci. Com.* 1889-95. *Trans.* 5.

†1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

Elected

1889 TURNBULL, GEORGE LINDSAY, M.B., Grove House, 76, Ladbroke grove.

1875 TURNER, FRANCIS CHARLEWOOD, M.D., Physician to the London Hospital; Consulting Physician to the North-Eastern Hospital for Children; 15, Finsbury square. C. 1895—.

1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital: 49, Green street, Park lane.

1894 TURNER, PHILIP DYMOCK, M.D., 95, Cromwell Road.

1896 TURNER, WILLIAM ALDREN, M.D., 13, Queen Anne street, Cavendish square.

1891 TWEED, REGINALD, M.D., 55, Upper Brook street, Grosvenor square.

1892 TWEEDY, JOHN, Professor of Ophthalmic Medicine and Surgery in University College, Ophthalmic Surgeon to University College Hospital, and to the Royal London Ophthalmic Hospital; 100, Harley Street, Cavendish square.

1876 VENN, ALBERT JOHN, M.D., 70A, Grosvenor street, and Hemnal Wood, Chislehurst.

1870 VENNING, EDGCOMBE, 30, Cadogan place.

†1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy, and Senior Physician to the French Hospital and Dispensary, Shaftesbury Avenue; 19A, Hanover square.

1891 VOELCKER, ARTHUR FRANCIS, M.D., B.S., Pathologist and Curator of the Museum, and Lecturer on Biology at the Middlesex Hospital; 31, Harley street.

1886 WAINEWRIGHT, BENJAMIN, M.B., C.M., Assistant Surgeon to the Royal Westminster Ophthalmic Hospital; 67, Grosvenor street, Grosvenor square.

Elected

1884 WAKLEY, THOMAS, jun., 5, Queen's Gate, South Kensington.

1883 WALLER, AUGUSTUS, M.D., F.R.S., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road, St. John's Wood. *Referee*, 1895—.

1888 WALLIS, FREDERICK CHARLES, M.B., B.C., Assistant Surgeon to the Charing Cross Hospital; 26, Welbeck street, Cavendish square.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Senior Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 77, Harley street, Cavendish square. C. 1888-9. *Referee*, 1895—. *Lib. Com.* 1882-5. *Trans.* 7.

1886 WARD, ALLAN OGIER, M.D., Lansdowne House, High road, Tottenham.

1890 WARD, ARTHUR HENRY, Surgeon to Out-patients, Lock Hospital; 7, Hertford street, Mayfair.

1891 WARING, HOLBURT JACOB, M.B., B.S., B.Sc., 9, Upper Wimpole street.

1877 WARNER, FRANCIS, M.D., Physician to, and Lecturer on Materia Medica and Therapeutics at, the London Hospital; 5, Prince of Wales terrace, Kensington Palace. *Trans.* 1.

1889 WASHBOURN, JOHN WYCHENFORD, M.D., Assistant Physician to, Physician in Charge of Electrical Department, Joint Lecturer on Physiology, and Demonstrator of Bacteriology at, Guy's Hospital; Physician to the London Fever Hospital; 15, Trinity square, S.E. *Trans.* 1.

1894 WATERHOUSE, HERBERT FURNIVALL, C.M., Assistant Surgeon to the Charing Cross Hospital; 81, Wimpole street.

†1861 WATSON, WILLIAM SPENCER, M.B., Surgeon to the Throat Department of the Great Northern Central Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.

Elected

1879 DE WATTEVILLE, ARMAND, M.D., 30, Welbeck street, Cavendish square.

1892 WEAVER, FREDERICK POYNTON, M.D., Cedar Lawn, Hampstead Heath.

†1891 WEBER, FREDERIC PARKES, M.D., Physician to the German Hospital, 19, Harley street, W.

1857 WEBER, HERMANN, M.D., Consulting Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Sci. Com.* 1889—. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73 *Trans.* 6.

1896 WEIR, ARTHUR NESHAM, M.B., 55, St. Charles square, Bayswater.

1895 WELLS, SYDNEY RUSSELL, M.B., 29, Devonshire street, Portland place.

†1854 WELLS, SIR THOMAS SPENCER, Bart., Hon. M.D. Dublin, Leyden, Bologna, Charkoff; Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Foreign Associate, Académie de Médecine, Paris; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 14. *Pro.* 1.

†1842 WEST, CHARLES, M.D., Foreign Associate, Académie de Médecine, Paris; 4, Evelyn Mansions, Carlyle place, Victoria street. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 WEST, SAMUEL, M.D., Assistant Physician to St. Bartholomew's Hospital; Senior Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square. C. 1894-5. *Lib. Com.* 1892—4. *Trans.* 5.

1888 WETHERED, FRANK JOSEPH, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 83, Harley street, Cavendish square. *Trans.* 1.

1881 WHARRY, ROBERT, M.D., 6, Gordon square.

Elected

1875 WHIPHAM, THOMAS TILLYER, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square. C. 1892-3.

1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 144, Sloane street.

1881 WHITE, WILLIAM HALE, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Referee*, 1888—. *Trans.* 4.

1890 WHITE-COOPER, W. G. O., M.B., 5, Courtfield road, Gloucester road, S.W.

1877 WHITMORE, WILLIAM TICKLE, Senior Surgeon to the Westminster General Dispensary, to the St. George's and St. James's Dispensary, and to the Gordon Hospital for Diseases of the Rectum; 7, Arlington street, Piccadilly.

1863 WILKS, SAMUEL, M.D., LL.D., F.R.S., Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street. *Referee*, 1872–81.

1890 WILLCOCKS, FREDERICK, M.D., Physician to Out-Patients, and Lecturer on Materia Medica and Therapeutics, at the Charing Cross Hospital; Physician to the Evelina Hospital for Sick Children; 14, Mandeville place, Manchester square.

†1865 WILLETT, ALFRED, *Trustee;* Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. V.P. 1890-91. *Referee*, 1882-89, 1892—. *Bldg. Com.* 1889-92. *Ho. Com.* 1892—. *Trans.* 2.

1887 WILLETT, EDGAR, M.B., 25, Welbeck street, Cavendish square.

1896 WILLIAMS, ALFRED HENRY, M.D., Harrow-on-the-Hill.

1888 WILLIAMS, CAMPBELL, 24, Welbeck street, Cavendish square.

Elected

1866 WILLIAMS, CHARLES THEODORE, M.D., *Trustee for Debenture-holders;* Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook street, Grosvenor square. C. 1884-5. *Referee,* 1888—. *Lib. Com.* 1880-3. *Sci. Com.* 1889—. *Trans.* 5.

1881 WILLIAMS, DAWSON, M.D., Physician to the East London Hospital for Children; 101, Harley street. *Trans.* 1.

1872 WILLIAMS, SIR JOHN, Bart., M.D., Physician-Accoucheur to H.R.H. the Princess Beatrice; Emeritus Professor of Obstetric Medicine, University College, London; Consulting Obstetric Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1891. *Referee,* 1878-90. *Lib. Com.* 1876-82.

1890 WILLS, WILLIAM ALFRED, M.D., Assistant Physician to the Westminster Hospital; Senior Physician to the North-Eastern Hospital for Children; 29, Lower Seymour street, Portman square.

1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.

1885 WOLFENDEN, RICHARD NORRIS, M.D., Physician to the Hospital for Diseases of the Throat, Golden square; 19, Harley street, Cavendish square.

1887 WOOD, THOMAS OUTTERSON, M.D., Senior Physician to the West End Hospital for Nervous Diseases; 40, Margaret street, Cavendish square.

1891 WOODFORDE, ALFRED POWNALL, 160, Goldhawk road.

1892 WOODHEAD, GERMAN SIMS, M.D., Director of the Research Laboratory, Conjoint Board of R.C.P.Lond. and R.C.S.Eng.; 1, Nightingale lane, Balham.

1890 WYNTER, WALTER ESSEX, M.D., Assistant Physician to the Middlesex Hospital; 30, Upper Berkeley street, Portman square.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION

1838 Henry Spencer Smith.
1840 Sir James Paget, Bt., F.R.S.
1841 Paul Jackson.
1842 Sir John Simon, K.C.B., F.R.S.
Charles West, M.D.
Sir John Erichsen, Bt., F.R.S.
1843 Henry Lee.
1845 George D. Pollock.
Sir Edwin Saunders.
Edward U. Berry.
1848 Sir Edward H. Sieveking, M.D.
John Clarke, M.D.
1849 C. H. F. Routh, M.D.
1850 Sir R. Quain, Bt., M.D., F.R.S.
1851 John Birkett.
John A. Kingdon.
Peter Y. Gowlland.
Bernard E. Brodhurst.
Robert J. Spitta, M.D.
1852 William Adams.
Sir Henry Thompson.
1853 Robert Brudenell Carter.
1854 Sir Alfred B. Garrod, M.D., F.R.S.
Sir Thomas Spencer Wells, Bt.
1855 William Marcet, M.D., F.R.S.
1856 Charles J. Hare, M.D.
William Bird.
Jonathan Hutchinson, F.R.S.
Timothy Holmes.
Alonzo H. Stocker, M.D.
1857 Sir William Overend Priestley, M.D.
George Harley, M.D., F.R.S.
Hermann Weber, M.D.
Henry Cooper Rose, M.D.
Henry Walter Kiallmark.
1858 John William Ogle, M.D.
1859 Wm. Howship Dickinson, M.D.
Edwin Thomas Truman.
Richard Barwell.
Edward Tegart.
1860 William Ogle, M.D.
Thomas Bryant.
John Couper.
Henry Howard Hayward.
1861 William Spencer Watson.
1862 Lionel Smith Beale, M.B., F.R.S.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D.
George Cowell.
1863 J. L. H. Langdon-Down, M.D.
Samuel Wilks, M.D., F.R.S.
Samuel Fenwick, M.D.
Julius Althaus, M.D.
Sydney Ringer, M.D., F.R.S.
Thomas Smith.
Arthur B. R. Myers.
William Sedgwick.
1864 John Harley, M.D.
Thomas William Nunn.
1865 James Edward Pollock, M.D.

1865 Reginald Southey, M.D.
George Fielding Blandford, M.D.
Sir Dyce Duckworth, M.D.
Frederick W. Pavy, M.D., F.R.S.
John Langton.
Frederick James Gant.
Alfred Willett.
Bowater John Vernon.
Alfred Cooper.
Christopher Heath.

1866 Thomas Fitz-Patrick, M.D.
Samuel Jones Gee, M.D.
Charles Theodore Williams, M.D.
Heywood Smith, M.D.
William Selby Church, M.D.

1867 Achille Vintras, M.D.
Richard Douglas Powell, M.D.
F. Howard Marsh.
Henry Power.
Sir William MacCormac.
Thomas Pickering Pick.
Charles Arthur Aikin.

1868 H. Charlton Bastian, M.D., F.R.S.
Sir W. H. Broadbent, Bart., M.D.
Thomas Buzzard, M.D.
John Cavafy, M.D.
Walter Butler Cheadle, M.D.
T. Henry Green, M.D.
William Chapman Grigg, M.D.
John Croft.
George Eastes.

1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.
Thomas Laurence Read.

1870 J. Warrington Haward.
Edgcombe Venning.
Clement Godson, M.D.
Reginald Harrison.
Robert Leamon Bowles, M.D.

1871 William Cayley, M.D.
T. Lauder Brunton, M.D., F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
Henry Sutherland, M.D.
George Vivian Poore, M.D.
Walter Rivington, M.S.
Benjamin Duke, M.D.

1872 T. Gilbart-Smith, M.D.
George B. Brodie, M.D.
Sir John Williams, Bart., M.D.
Sir J. Fayrer, M.D., F.R.S.
Charles S. Tomes, M.A., F.R.S.
Sir William Bartlett Dalby.

1873 William Miller Ord, M.D.
Frederick Taylor, M.D.
Norman Moore, M.D.
John Curnow, M.D.
William R. Gowers, M.D., F.R.S.
Sir Wm. Guyer Hunter, M.D.
Jeremiah McCarthy.
Wm. Johnson Smith.
Robert William Parker.
Alex. O. MacKellar.
Henry T. Butlin.
Charles Higgens.
William J. Walsham.

1874 Alfred Lewis Galabin, M.D.
George Thin, M.D.
John Mitchell Bruce, M.D.
Henry Morris.
William Laidlaw Purves.
William Harrison Cripps.
Henry G. Howse, M.S.
Herbert William Page.
Frederic Durham.
William Robert Smith, M.D.

1875 Thomas T. Whipham, M.D.
Francis Charlewood Turner, M.D.
Thomas Crawford Hayes, M.D.
Charles Henry Carter, M.D.
Waren Tay.
Edmund J. Spitta.
Samuel C. Osborn.
Fletcher Beach, M.B.

1876 Thomas Barlow, M.D.
Wm. Lewis Dudley, M.D.
Albert J. Venn, M.D.
John Knowsley Thornton, M.B.
N. Charles Macnamara.
John N. C. Davies-Colley, M.C.

1877 Felix Semon, M.D.
Sidney Coupland, M.D.
Francis Warner, M.D.
William Ewart, M.D.
Alfred Pearce Gould, M.S.
Rickman J. Godlee, M.S.
Alban H. G. Doran.
George Ernest Herman, M.B.
Samuel West, M.D.
John Abercrombie, M.D.
George Allan Heron, M.D.
Joseph A. Ormerod, M.D.
P. Henry Pye-Smith, M.D., F.R.S.
Edward Nettleship.
William Henry Bennett.
William T. Whitmore.

1878 Sir Jas. Crichton-Browne, M.D.
Fred. T. Roberts, M.D.
Sir Joseph Lister, Bart., F.R.S.

1878 Clinton T. Dent.
John H. Morgan.
Donald W. Charles Hood, M.D.
1879 Edward Woakes, M.D.
Armand de Watteville, M.D.
Malcolm A. Morris.
A. E. Cumberbatch.
Edmund Owen.
Arthur E. J. Barker.
Frederick Treves.
Horatio Donkin, M.D.
Thomas John Maclagan, M.D.
Andrew Clark.
Francis Henry Champneys, M.D.
William Watson Cheyne, F.R.S.
George Henry Savage, M.D.
H. H. Clutton, M.A.
Frederic S. Eve.
E. Noble Smith.
William Henry Allchin, M.D.
F. G. Dawtrey Drewitt, M.D.
1880 Robert Alex. Gibbons, M.D.
David Ferrier, M.D., F.R.S.
Vincent Dormer Harris, M.D.
Edmund Distin Maddick.
Jas. John MacWhirter Dunbar, M.D.
James William Browne, M.B.
William Appleton Meredith, M.B.
Malcolm Macdonald McHardy.
A. Boyce Barrow.
William Murrell, M.D.
Leslie Ogilvie, M.B.
George Ogilvie, M.B.
Charles Edward Beevor, M.D.
Thomas Colcott Fox, M.B.
George Henry Makins.
1881 Francis de Havilland Hall, M.D.
Robert Wharry, M.D.
Cecil Yates Biss, M.D.
Richard Clement Lucas, B.S.
Stephen Mackenzie, M.D.
William Hale White, M.D.
Eustace Smith, M.D.
Percy Kidd, M.D.
Oswald A. Browne.
W. Bruce Clarke, M.B.
Dawson Williams, M.D.
George Lindsay Johnson, M.D.
Henry Edward Juler.
Jonathan F. C. H. Macready.
C. B. Lockwood.
1882 Philip J. Hensley, M.D.
Ernest Clarke, M.D.
John Barclay Scriven.
1882 George Robertson Turner.
Howard Henry Tooth, M.D.
Herbert Isambard Owen, M.D.
Charles R. B. Keetley.
Anthony A. Bowlby.
Amand J. McC. Routh, M.D.
Seymour J. Sharkey, M.D.
William Lang.
Henry Radcliffe Crocker, M.D.
1883 Edwin Clifford Beale, M.A., M.B.
James Kingston Fowler, M.D.
James Frederic Goodhart, M.D.
John Charles Galton, M.A.
W. Hamilton A. Jacobson, M.Ch.
Walter H. Jessop, M.B.
Walter Edmunds, M.C.
Victor A. Horsley, F.R.S.
Dudley Wilmot Buxton, M.D.
Charles Douglas F. Phillips, M.D.
John James Pringle, M.B.
Henry Roxburgh Fuller, M.D.
Wilmot Parker Herringham, M.D.
Augustus Waller, M.D.
William Pasteur, M.D.
Edward Albert Schäfer, F.R.S.
John Bland Sutton.
William Rose, M.B.
Storer Bennett.
Robert Marcus Gunn, M.B.
James Dixon Bradshaw, M.B.
1884 George Newton Pitt, M.D.
Charles Stonham.
Stanley Boyd, M.B.
William Arbuthnot Lane, M.S.
Arthur Marmaduke Sheild, M.B.
Frederic Bowreman Jessett.
Sidney Harris Cox Martin, M.D.
George Lawson.
Thomas Wakley, jun.
F. Swinford Edwards.
James Johnston, M.D.
William Duncan, M.D.
Charles Chinner Fuller.
Jean Samuel Keser, M.D.
George Richard Turner Phillips.
Bilton Pollard.
1885 Alexander Haig, M.D.
Theodore Dyke Acland, M.D.
Frederick Walker Mott, M.D.
James Berry.
John Cahill.
John Poland.
Heinrich Port, M.D.
R. Norris Wolfenden, M.D.

1885 A. C. Butler-Smythe.
Charles Alfred Ballance, M.S.
Walter S. A. Griffith, M.D.
John Edward Squire, M.D.
John D. Malcolm, M.B., C.M.
Phineas S. Abraham, M.D.
Henry Willingham Gell, M.B.
1886 Robert Maguire, M.D.
Harrington Sainsbury, M.D.
Cuthbert Hilton Golding-Bird, M.B.
Benjamin Wainewright, M.B., C.M.
Charles E. Leopold B. Hudson.
Lauriston Elgie Shaw, M.D.
Charters James Symonds, M.S.
Robert Boxall, M.D.
Allan Ogier Ward, M.D.
Archibald Edward Garrod, M.D.
Stephen Paget.
William Radford Dakin, M.D.
Samuel Herbert Habershon, M.D.
Arthur Quarry Silcock.
Arthur H. N. Lewers, M.D.
1887 Walter George Spencer.
Thomas Outterson Wood, M.D.
Edgar William Willett, M.B.
Henry Lewis Jones, M.D.
Francis George Penrose, M.D.
Hugh Percy Dunn.
Frederic William Hewitt, M.D.
Harry Scott, M.D.
James Barry Ball, M.D.
Gilbert Richardson, M.D.
D'Arcy Power, M.B.
Charles Arkle, M.D.
John Gay.
James Calvert, M.D.
Percy J. F. Lush, M.B.
1888 Robert Henry Scanes Spicer, M.D.
Jonathan Hutchinson, jun.
Campbell Williams.
James Donelan, M.B., C.M.
John Anderson, M.D., C.I.E.
Laurie Asher Lawrence.
Arthur Pearson Luff, M.D., B.Sc.
Albert Carless, M.B., B.S.
Frederick C. Wallis, M.B., B.C.
Charles James Cullingworth, M.D.
Edmund Cautley, M.D., B.C.
H. Montague Murray, M.D.
Arthur Symons Eccles, M.B.
Frank Joseph Wethered, M.D.
Edmund Wilkinson Roughton, M.D.
Frederick William Cock, M.D.
John Phillips, M.D.
1889 Montagu Handfield-Jones, M.D.
Norman M. MacLehose, M.B.
David Henry Goodsall.
Raymond Johnson, M.B.
John Fletcher Little, M.B.
Henry Work Dodd.
George Lindsay Turnbull, M.B.
Sir William Roberts, M.D., F.R.S.
Sidney Phillips, M.D.
William Charles Bull, M.B.
George P. Field.
John Wychenford Washbourn, M.D.
Charles Henry Cosens.
Henry Percy Dean, M.B., M.S.
Alfred Samuel Gubb.
William Hunter, M.D.
J. Inglis Parsons, M.D.
Bernard Pitts, M.B., M.C.
Robert Percy Smith, M.D., B.S.
Herbert R. Spencer, M.D., B.S.
Nestor Isidore Chas. Tirard, M.D.
1890 John Rose Bradford, M.D., F.R.S.
Roland Danvers Brinton, M.D.
James Cagney, M.D.
Charles D. B. Hale, M.D.
Edwin Cooper Perry, M.D.
Morton Smale.
Frederick Willcocks, M.D.
R. Ashton Bostock.
William T. Holmes Spicer, M.B.
Thomas Henry Crowle.
Henry Walter Syers, M.D.
Seymour Taylor, M.D.
William Alfred Wills, M.D.
G. O. White-Cooper, M.B.
Herbert William Allingham.
William Anderson.
William A. F. Bateman.
James Jackson Clarke, M.B.
Leonard G. Guthrie, M.B., B.S.
G. William Hill, M.D., B.Sc.
Edward Law, M.D., C.M.
Patrick Manson, M.D., C.M.
William Wallis Ord, M.D.
Humphry D. Rolleston, M.D., B.C.
Arthur Henry Ward.
Walter Essex Wynter, M.D., B.S.
1891 William Lee Dickinson, M.D.
Herbert P. Hawkins, M.D., B.C.
Cyril Ogle, M.A., M.B.
Leonard Remfry, M.D.
Arthur F. Voelcker, M.D., B.S.
Alfred Pownall Woodforde.
Charles Gordon Brodie.

1891 Herbert T. Herring, M.B., B.S.
Ernest Muirhead Little.
Henry Charrington Martin, M.D.
Frederick William Andrewes, M.B.
Alfred Eddowes, M.D.
Herbert Morley Fletcher, M.D.
William Heaton Hamer, M.D.
William Bromfield Paterson.
Reginald Tweed, M.D.
Holburt Jacob Waring.
Frederic Parkes Weber, M.D.
F. E. Batten, M.D.
Thomas Jessopp Bokenham.
Norman Dalton, M.D.
P. R. W. De Santi.
P. W. Dove.
William J. Gow, M.D.
Charles Arthur Mercier, M.B.
Paul Frank Moline, M.B.
Edward Percy Paton, M.D.
Arthur Bowen Rendel, M.B., B.C.
M. Armand Ruffer, M.D.
James Samuel Risien Russell, M.B.
George Cockburn Smith.
Charles Percival White, M.B., B.C.
W. Page May, M.D.
Richard J. Reece.

1892 Edward Cotterell.
J. Dundas Grant, M.D.
R. J. Bliss Howard, M.D.
Thomas Horrocks Openshaw, M.B.
Harry Marmaduke Page.
William Bezly Thorne, M.D.
German Sims Woodhead, M.D.
W. H. Russell Forsbrook, M.D.
John Harold.
William Ward Leadam, M.D.
John Alfred Masters, M.D.
Gustave Schorstein, M.B.
Charles Sempill de Segundo.
John Tweedy.
E. H. Myddelton-Gavey.
E. Matthews James.
J. S. Selwyn-Harvey, M.D.
St. Clair Thomson, M.D.
F. Manley B. Sims.
Solomon Charles Smith, M.D.
F. Poynton Weaver, M.D.
Henry Rayner, M.D.

1893 James Taylor, M.D.
Howard Barrett.
Robert Cozens Bailey, M.B.
Henry Albert Caley, M.D.
Arthur Edward Giles, M.D.
Miles Miley, M.B.
Alfred A. Kanthack, M.D.
Kenneth McLeod, M.D.
D. Watkin Roberts, M.D.
Leonard A. Bidwell.
Frédéric F. Burghard, M.D., M.S.
William McAdam Eccles, M.S.
Vaughan Harley, M.D.
George Herschell, M.D.
Arnold Lawson.
Walter Knowsley Sibley, M.D.
Richard Sisley, M.D.

1894 Richard Gill.
Joseph Sefton Sewill.
Thomas Vincent Dickinson, M.D.
Herbert Edward Durham, M.B.
Alexander Morison, M.D.
L. Hemington Pegler, M.D.
Herbt. Furnivall Waterhouse, M.D.
Philip D. Turner, M.D.
Percy Furnivall.
R. L. Langdon-Down, M.B., B.C.
Allan Macfadyen, M.D., B.S.
Ernst Michels, M.D.
Wm. Rivers Pollock, M.B., B.C.
Thomas R. Smith, M.B., B.C.
Charles Slater, M.B.

1895 Charles Arthur Parker.
Sydney Russell Wells, M.B.
Alfred Milne Gossage, M.B.
Robert Murray Leslie, M.B.
Gerald R. Baldwin.
James Galloway, M.D.
E. E. Henderson, M.B., B.C.
Daniel John Leech, M.D.
David Bridge Lees, M.D.
Arthur Newsholme, M.D.
Arthur G. Phear, M.D.

1896 Joseph Lockhart Downes, M.B.
Edward Wilberforce Goodall, M.D.
James Ernest Lane.
George Oliver, M.D.
George Alex. Sutherland, M.D.
Charles F. Buttar, M.B.
P. J. Freyer, M.D., I.M.S., M.A.
Percival Horton-Smith, M.B.
William Edward Lee, M.B.
Frederick Henry Lewis, M.B.
James Keogh Murphy, M.B.
Thomas William Shore, M.D.
John Stretton Sloane, M.B.
William Aldren Turner, M.D.
Arthur Nesham Weir, M.B.
Alfred Henry Williams, M.D.

1896 Frank Belben, M.B.
John Brian Christopherson, M.B.
Surg.-Capt. Matthew L. Hughes
Samuel Hyde, M.D.

1896 John William Moore, M.D.
Charles Hubert Roberts, M.D.
John W. Watson Stephens, M.B.
Charles R. J. Atkin Swan, M.B.

NON-RESIDENT FELLOWS

Elected

*1851 *ACLAND, SIR HENRY W.*, Bart., K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; late Regius Professor of Medicine in the University of Oxford; Oxford.

1839 ALCOCK, SIR RUTHERFORD*, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD*, M.D., LL.D.Glasgow, F.R.S., Regius Professor of Physic, Univ. Camb.; Consulting Physician to the Leeds General Infirmary; St. Rhadegund's, Cambridge. *Trans.* 3.

1884 ANDERSON, ALEXANDER RICHARD*, Surgeon to the General Hospital, 5, East Circus Street, Nottingham. *Trans.* 1.

1862 ANDREW, JAMES*, M.D., Branksome avenue, West Bournemouth. S. 1878-9. C. 1881-2. V.P. 1888. *Trans.* 1.

*1880 *APPLETON, HENRY*, M.D., 2, Swinburn villas, Romford, Essex.

*1873 *BAKER, J. WRIGHT*, Consulting Surgeon to the Derbyshire General Infirmary [care of Dr. Benthall, 101, Friar gate, Derby].

1891 BALGARNIE, WILFRED*, M.B., The Dutch House, Hartley Wintney, Winchfield.

†*1848* BALLARD, EDWARD*, M.D., F.R.S., late Inspector, Medical Department, Local Government Board; 6, Ravenscroft Park, High Barnet, Herts. C. 1872. V.P. 1875-6. *Sci. Com.* 1889—. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

Elected

*1866 *BANKS, SIR JOHN*, M.D., LL.D., D.Sc., K.C.B., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to Sir Patrick Dun's and City of Dublin Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Royal University in Ireland; 45, Merrion square, Dublin.

1886 *BANKS, WILLIAM MITCHELL*, M.D., Surgeon to the Liverpool Royal Infirmary 28, Rodney street, Liverpool.

1882 *BARKER, FREDERICK CHARLES*, M.D., Surgeon-Major, Bombay Medical Service.

*1881 *BARNES, HENRY*, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

†1861 *BARNES, ROBERT*, M.D., Liss, Hants. C. 1877-8. V.P. 1889–90. *Referee*, 1867-76, 1891—. *Lib. Com.* 1869-73. *Sci. Com.* 1889—. *Trans.* 4.

1840 *BARROW, BENJAMIN*, Consulting Surgeon to the Royal Isle of Wight Infirmary; Southlands Park road, Ryde.

*1860 *BEALEY, ADAM*, M.D., M.A., Filsham Lodge, Filsham road, St. Leonard's-on-Sea, Sussex.

1856 *BEARDSLEY, AMOS*, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1880 *BENNETT, ALEXANDER HUGHES*, M.D. (Travelling).

1889 *BENTLEY, ARTHUR J. M.*, M.D., Mena House, Pyramids, Cairo, Egypt.

1872 *BEVERLEY, MICHAEL*, M.D., Surgeon to the Norfolk and Norwich Hospital; 54, Prince of Wales road, Norwich.

*1865 *BICKERSTETH, EDWARD ROBERT*, Consulting Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool. *Trans.* 1.

1892 *BICKERSTETH, ROBERT ALEXANDER*, M.A., M.B., Assistant Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool.

1878 *BINDON, WILLIAM JOHN VEREKER*, M.D., 48, St. Ann's street, Manchester.

Elected

†1849 *Birkett, Edmund Lloyd*, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; Westbourne Rectory, Emsworth, Hampshire. C. 1865-6. *Referee*, 1851-9.

1865 *Blanchet, Hilarion*, Examiner to the College of Physicians and Surgeons, Lower Canada; 35, Conillard street, Quebec, Canada.

1869 *Bourne, Walter*, M.D. (Travelling).

1874 *Bradshaw, A. F.*, C.B., Surgeon Major-General, Principal Medical Officer, H.M.'s Forces in India; Simla, India. [Agents: Holt & Co., 17, Whitehall place.]

1876 *Bridges, Robert*, M.B., Manor House, Yattendon, Newbury, Berks.

1867 *Bridgewater, Thomas*, M.B., LL.D., Harrow-on-the-Hill, Middlesex.

1892 *Bronner, Adolph*, M.D., 33, Manor row, Bradford.

1894 *Brook, William Henry Breffit*, 1, James street, Lincoln.

1888 *Browne, Henry Langley*, Moor House, West Bromwich.

1881 *Browne, John Walton*, M.D., Surgeon to the Belfast Royal Hospital; Surgeon to the Belfast Ophthalmic Hospital; 10, College square N., Belfast.

1864 *Buckle, Fleetwood*, M.D., Merton Lodge, Merton road, Southsea.

1871 *Butt, William F.*

*1851 *Cadge, William*, Consulting Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.

1891 *Campbell, Henry Johnstone*, M.D., 157, Manningham lane, Bradford.

1888 *Carter, William Jeffreys Becher*, Aliwal North, Cape Colony.

Elected

1884 CHAFFEY, WAYLAND CHARLES, M.D., Physician to the Royal Alexandra Hospital for Children; 13, Montpellier road, Brighton.

1859 CHANCE, FRANK, M.B., Burleigh House, Sydenham hill.

1885 CHAPMAN, PAUL MORGAN, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.

**1881* CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; Consulting Surgeon to the Bromsgrove Hospital; 22, Temple row, Birmingham. *Trans.* 3.

**1873* CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

1892 CLARK, JAMES CHARLES, 35, Castle road, Bedford.

1888 CLARKE, ROBERT HENRY, M.B., Bellevue, Lympne, Hythe, till end of 1896.

1857 COATES, CHARLES, M.D., Consulting Physician to the Bath Royal United Hospital; 10, Circus, Bath.

1868 COCKLE, JOHN, A.M., M.D., F.L.S., Consulting Physician to the Royal Free Hospital; The Lodge, West Molesey. *Trans.* 2.

1893 COLE, ROBERT HENRY, M.D., Moorcroft, Hillingdon, Uxbridge.

1891 COOK, HERBERT GEORGE, M.D., B.S., 22, Newport road, Cardiff.

1891 COUMBE, JOHN BATTEN, M.D., Rosslyn, Clevedon, Somerset.

1894 CRAWFORD, WILLIAM SMYTH, Assistant Surgeon to the Liverpool Cancer and Skin Hospital; 77, Mount Pleasant, Liverpool.

**1869* CRESSWELL, PEARSON R., Surgeon to the Merthyr General Hospital; Dowlais, Merthyr Tydvil.

Elected

1892 *Cross, Francis Richardson*, M.B., Ophthalmic Surgeon to the Bristol Royal Infirmary, and Surgeon to the Bristol Eye Hospital; Worcester House, Clifton, Bristol.

1895 *Dardel, Jean*, M.D., Aix-les-Bains, Savoy.

1879 *Darwin, Francis*, M.B., F.R.S., Wychfield, Huntingdon road, Cambridge.

1874 *Davidson, Alexander*, M.D., Physician to the Liverpool Royal Infirmary; 2, Gambier terrace, Liverpool.

1878 *Davy, Richard*, Consulting Surgeon to the Westminster Hospital; Burstone House, Bow, North Devon. *Trans.* 1.

1882 *Dawson, Yelverton*, M.D., Heathlands, Southbourne-on-Sea, Hants.

1889 *Delépine, Sheridan*, B.S., M.B., Professor of Pathology Owens College, Manchester. *Trans.* 1.

1867 *Drage, Charles*, M.D., Hatfield, Herts.

1884 *Drage, Lovell*, M.D., B.Ch.Oxon., Burleigh Mead, Hatfield, Herts.

1885 *Drummond, David*, M.D., 7, Saville place, Newcastle-on-Tyne.

1880 *Drury, Charles Dennis Hill*, M.D., Bondgate, Darlington.

1871 *Dukes, Clement*, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.

1867 *Dukes, Major Charles*, M.D., Clarence Villa, Torrs park, Ilfracombe.

1889 *Duncan, John*, M.D., St. Petersburg, Russia.

1843 *Durrant, Christopher Mercer*, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.

1872 *Eager, Reginald*, M.D., Northwoods, near Bristol.

Elected

1887 *Easmon, John Farrell*, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony; Accra, Gold Coast, West Africa.

1887 *Elliott, John*, Whitefriars Lodge, Chester.

1848 *Ellis, George Viner*, Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

1868 *Ellis, James*, M.D., The Sanatorium, Anaheim, Los Angeles County, California.

*1854 *Ellison, James*, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1889 *Elliston, William Alfred*, M.D., Stoke Hall, Ipswich.

*1875 *Fagan, John*, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital; 19, Great Victoria street, Belfast.

1869 *Fairbank, Frederick Royston*, M.D., 3, Eversfield place, St. Leonard's-on-Sea.

1887 *Feeny, Michael Henry*, Les Avants, Montreux, Switzerland.

*1872 *Fenwick, John C. J.*, M.D., Physician to the Durham County Hospital; Long Framlington, Morpeth.

1879 *Finlay, David White*, M.D., Professor of the Practice of Medicine in the University of Aberdeen; Physician to the Aberdeen Royal Infirmary; Consulting Physician to the Royal Hospital for Diseases of the Chest, London; 2, Queen's terrace, Aberdeen. *Referee*, 1891-3. *Trans.* 2.

1842 *Fletcher, Thomas Bell Elcock*, M.D., Consulting Physician to the Birmingham General Hospital; 8, Clarendon crescent, Leamington. *Trans.* 1.

*1864 *Folker, William Henry*, Consulting and late Hon. Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1892 *Foster, Michael George*, M.A., M.B., Great Shelford, Cambridge.

1859 *Fox, Edward Long*, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.

Elected

1871 *Frank, Philip*, M.D., Cannes, France.

**1884* *Franks, Kendal*, M.D., Surgeon to the Adelaide Hospital and to the Throat and Ear Hospital, Dublin; Surgeon in Ordinary to the Lord Lieutenant; 6, Fitzwilliam square, Dublin. *Trans.* 2.

**1889* *Freeman, Henry William*, 24, The Circus, Bath.

1876 *Furner, Willoughby*, M.D., Surgeon to the Sussex County Hospital; Brunswick square, Brighton.

**1864* *Gairdner, William Tennant*, M.D., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow. *Trans.* 1.

1885 *Gamgee, Arthur*, M.D., F.R.S., Montreux, Switzerland.

1867 *Garland, Edward Charles*, Yeovil, Somerset.

1867 *Garlike, Thomas W.*, Malvern Cottage, Churchfield road, Ealing.

1879 *Garstang, Thomas Walter Harrop*, Headingley House, Knutsford, Cheshire.

**1889* *Gaskell, Walter Holbrook*, M.D., F.R.S., Lecturer on Physiology, University of Cambridge; The Uplands, Great Shelford, Cambs.

1884 *Gibbes, Heneage*, M.D., Professor of Pathology in the University of Michigan; Ann Arbor, Michigan, U.S.A.

1893 *Gordon, William*, M.B., M.C.

**1890* *Gordon, William*, M.D., Barnfield Lodge, Exeter.

1889 *Greene, George Edward Joseph*, Monte Vista, Ferns, County Wexford.

**1875* *Greenfield, William Smith*, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.

1882 *Gresswell, Dan Astley*, M.D., D.P.H., Melbourne, Victoria.

[illegible]

[illegible] *Griffiths, Joseph*, M.A., M.D., C.M., Assistant to the Professor of Surgery in the University of Cambridge; 4, King's parade, Cambridge.

[illegible] *Gruby, John*, Pitt House, 15, Johnstown street, Bath.

[illegible] *Hamilton, Robert*, Consulting Surgeon to the Royal Southern Hospital, Liverpool; Magheraybuoy, Portrush, Co. Antrim, Ireland.

[illegible] *Harnant, William Henry*, The Tower House, Clifton, Bristol.

[illegible] *Haviland, Alfred*, Douglas, Isle of Man.

[illegible] *Haviland, Frank Papillon*, M.B., B.C., 57, Warrior square, St. Leonard's-on-Sea.

[illegible] *Hawkins, Francis Henry*, M.B., Physician to the Royal Berkshire Hospital; 26, Portland place, Reading.

1861 *Hayward, William Henry*, Oxford road, Burnley, Lancashire.

†*1843* *Holden, Luther*, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1894 *Holland, James Frank*, M.D., St. Moritz, Engadine, Switzerland.

1868 *Hollis, William Ainslie*, M.D., Physician to the Sussex County Hospital; 8, Cambridge road, Brighton. *Trans.* 1.

†*1846* *Holthouse, Carsten*, 1, Bath terrace, Richmond. C. 1863. *Referee*, 1870-6. *Lib. Com.* 1859-60.

1895 *Howard, Benjamin*, M.D. [New York, U.S.A.] *Trans.* 1.

1881 *Howard, Henry*, M.B., Medical Officer of Health, Williamstown, Melbourne, Victoria.

1896 *Hughes, Matthew Louis*, Surg.-Capt., A.M.S., Valetta, Malta. *Trans.* 1.

1855 *Humphry, Sir George Murray*, M.D., D.Sc., LL.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 9.

1882 *Humphry, Laurence*, M.D., 3, Trinity street, Cambridge.

Elected

1849 *Hussey, Edward Law*, 24, Winchester road, Oxford. *Trans.* 1.

1896 *Hyde, Samuel*, M.D., Lismore House, 3, Hardwick street, Buxton.

1847 *Image, William Edmund*, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.

1863 *Jackson, Thomas Vincent*, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; Whetstone House, Waterloo road south, Wolverhampton.

**1883* *Jenkins, Edward Johnstone*, M.D., The Australian Club, Sydney, New South Wales.

†*1851* *Jenner, Sir William*, Bart., M.D., G.C.B., D.C.L., LL.D.Cantab., LL.D.Edin., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Greenwood, Bishop's Waltham, Hants. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

1881 *Jennings, William Oscar*, M.D., 35, Rue Marbœuf, Avenue des Champs-Elysées, Paris.

1851 *Johnson, Edmund Charles*, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

1889 *Johnson, Harold J.*, Senior Assistant, Gloucester County Asylum, Gloucester.

1848 *Johnstone, Athol Archibald Wood*, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1876 *Jones, Leslie Hudson*, M.D., Limefield House, Cheetham hill, Manchester.

**1875* *Jones, Philip Sydney*, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, and Fellow of the Senate, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., Wool Exchange, Coleman Street, E.C.]

Elected

1865 *Jordan, Furneaux*, Consulting Surgeon to the Queen's Hospital, Birmingham; Selly Hill, Birmingham.

1872 *Kelly, Charles*, M.D., Professor of Hygiene in King's College, London; Medical Officer of Health for the West Sussex Combined Sanitary District; Ellesmere, Gratwicke road, Worthing, Sussex.

**1848* *Kendell, Daniel Burton*, M.B., Thornhill House, Walton, near Wakefield, Yorkshire.

**1890* *Kerr, J. G. Douglas*, M.B., C.M., 6, The Circus, Bath.

**1877* *Khory, Rustomjee Naserwanjee*, M.D., Hormazd Villa, Khumballa hill, Bombay.

1888 *Kynsey, William Raymond*, C.M.G., Inspector-General of Hospitals, Colombo, Ceylon.

1889 *Lancaster, Ernest le Cronier*, M.B., B.Ch., Assistant Physician to the Swansea Hospital; Winchester House, Swansea, S. Wales.

**1873* *Larcher, O.*, M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1862 *Latham, Peter Wallwork*, M.D., late Downing Professor of Medicine, Cambridge University; Senior Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

**1890* *Lawrie, Edward*, M.B., Surgeon Lieutenant-Colonel, Indian Medical Department; Residency Surgeon; Hyderabad, Deccan.

1880 *Laycock, George Lockwood*, M.B., C.M., Melbourne, Victoria, Australia.

1892 *Lazarus-Barlow, Walter Sydney*, M.B., 17, Chesterton road, Cambridge. *Sci. Com.* 1892—.

**1886* *Lediard, Henry Ambrose*, M.D., Surgeon to the Cumberland Infirmary; 35, Lowther street, Carlisle. *Trans.* 1.

Elected

1882 *Ledwich, Edward l'Estrange*, Anatomist to the Royal College of Surgeons, Ireland; 31, Harcourt street, Dublin.

1895 *Leech, Daniel John*, M.D., Elm House, Whalley Range, Manchester. *Sci. Com.* 1896—.

1883 *Leeson, John Rudd*, M.D., C.M., Clifden House, Twickenham.

1869 *Legg, John Wickham*, M.D. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.

**1872* *Little, David*, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; Ophthalmic Surgeon to the Manchester Royal Infirmary; Lecturer on Ophthalmology at the Victoria University; 21, St. John street, Manchester.

**1889* *Little, James*, M.D., Physician to the Adelaide Hospital; Consulting Physician to the Rotunda, St. Mark's, Steevens', and the Children's Hospitals; 14, Stephen's Green North, Dublin.

1871 *Little, Louis Stromeyer*, Shanghai, China.

1894 *Lowe, Thomas Pagan*, 16, The Circus, Bath.

1883 *Lund, Edward*, Emeritus Professor of Surgery, Victoria University, Manchester; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.

1867 *Maberly, George Frederick*, Mailai Valley, Nelson, New Zealand.

1889 *MacAlister, Donald*, B.Sc., M.D., Physician to Addenbrooke's Hospital; Lecturer on Medicine, St. John's College; University Lecturer in Medicine; St. John's College, Cambridge.

1887 *Macdonald, George Childs*, M.D.

1866 *Macgowan, Alexander Thorburn*, M.D.

**1859* *M'Intyre, John*, M.D., LL.D., Odiham, Hants.

1876 *Mackey, Edward*, M.D., Senior Physician to the Royal Alexandra Hospital for Sick Children; Assistant Physician to the Sussex County Hospital; 3, Portland place, Brighton.

Elected

*1854 *MACKINDER, DRAPER*, M.D., Consulting Surgeon to the Dispensary; The Cedars, Gainsborough, Lincolnshire.

1891 *MANBY, ALAN REEVE*, M.D., Surgeon Apothecary to their Royal Highnesses the Prince and Princess of Wales at Sandringham; East Rudham, Norfolk.

1894 *MARRIOTT, CHARLES WILLIAM*, M.D., Aubrey House, Bath road, Reading.

1892 *MARTIN, CHRISTOPHER*, M.B., C.M., 35, George road, Edgbaston, Birmingham.

1883 *MAUDSLEY, HENRY CARR*, M.D., 22, Collins street, Melbourne, Victoria.

1839 *MEADE, RICHARD HENRY*, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1874 *MERRIMAN, JOHN J.*, 13, The Steyne, Worthing.

1895 *MILLS-ROBERTS, ROBERT HERBERT*, Hafod-ty, Llanberis, North Wales.

1887 *MIVART, FREDERICK ST. GEORGE*, M.D., Beaumont Lodge, Worple road, Wimbledon.

1896 *MOORE, JOHN WILLIAM*, M.D., M.Ch., 40, Fitzwilliam square west, Dublin.

1891 *MORRIS, GRAHAM*, Wallington, Surrey.

1894 *MORSE, THOMAS HERBERT*, 10, Upper Surrey street, Norwich. *Trans.* 1.

1873 *MURRAY, J. IVOR*, M.D., 24, Huntriss row, Scarborough.

1881 *NALL, SAMUEL*, M.B., Dryhurst Lodge, Disley, Stockport.

1889 *NAPIER, FRANCIS HORATIO*, M.B., Cape Town.

1870 *NEILD, JAMES EDWARD*, M.D., Lecturer on Forensic Medicine and Psychological Medicine in the University of Melbourne; 21, Spring street, Melbourne, Victoria.

1895 *NEWSHOLME, ARTHUR*, M.D., 11, Gloucester place, Brighton.

1868 *NICHOLLS, JAMES*, M.D., Trenarren, Newquay, Cornwall.

1849 *NORMAN, HENRY BURFORD*, The Manor-house, Drayton, Taunton, Somerset. *Lib. Com.* 1857.

*1847 *NOURSE, WILLIAM EDWARD CHARLES*, Bouverie House, Exeter.

Elected

1884 *Oakes, Arthur*, M.D., Warialda, Portarlington road, Bournemouth.

1880 *O'Connor, Bernard*, A.B., M.D., Physician to the North London Hospital for Consumption; 25, Hamilton road, Ealing.

1896 *Ogle, John Gilbert*, M.D., South Redlands, Reigate.

**1855* *Ogle, William*, M.D., late Physician to the Derbyshire Infirmary; The Elms, Duffield road, Derby.

1870 *Oldham, Charles Frederic*, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

**1883* *Oliver, Thomas*, M.A., M.D., Professor of Physiology, University of Durham; and Physician to the Newcastle-on-Tyne Infirmary; 7, Ellison place, Newcastle-on-Tyne. *Trans.* 1.

**1871* *O'Neill, William*, M.D., late Physician to the Lincoln Lunatic Hospital, 2, Lindum road, Lincoln.

1890 *Ord, William Wallis*, M.D., The Hall, Salisbury.

1885 *Ormsby, L. Hepenstal*, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.

1887 *Paget, Charles Edward*, Medical Officer of Health for the County Borough of Salford; Lecturer on Public Health, Owens College, Victoria University; North Bentcliffe, Eccles, Lancashire.

**1858* *Paley, William*, M.D., Physician to the Ripon Dispensary; Yore Bank, Ripon, Yorkshire.

1887 *Pardington, George Lucas*, M.D., 47, Mount Pleasant road, Tunbridge Wells.

1885 *Parker, Rushton*, M.B., B.S., Professor of Surgery, University College, Liverpool (Victoria University); Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.

1891 *Parkin, Alfred*, M.S., M.D., 5, Albion street, Hull. *Trans.* 1.

1879 *Peel, Robert*, 120, Collins street east, Melbourne, Victoria.

Elected

*1855 PEMBERTON, OLIVER, Consulting Surgeon to the Birmingham General Hospital, President and Emeritus Professor of Surgery at Mason's College, Birmingham; H.M. Coroner, Birmingham; 65, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, The Cedars, Broadwas-on-Teme, Worcester.

*1879 PESIKAKA, HORMASJI DOSABHAI, 43, Hornby road, Bombay.

1895 PHEAR, ARTHUR G., M.D., 28, Banbury road, Oxford.

*1878 PHILIPSON, GEORGE HARE, M.D., D.C.L., Professor of Medicine in Durham University; Senior Physician to the Newcastle-upon-Tyne Royal Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1891 PIERCE, BEDFORD, M.D., The Retreat, York.

†1841 PITMAN, SIR HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital; Cranbrook, Bycullah park, Enfield. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.

1892 POWELL, HERBERT ANDREWS, M.A., M.D., M.Ch., 9, St. Thomas's street, Winchester.

1857 VON RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1893 RANKIN, GUTHRIE, M.D., 23, Jury street, Warwick.

1890 RANSOM, WILLIAM BRAMWELL, M.D., Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Consulting Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.

1882 REID, SIR JAMES, M.D., K.C.B., Resident Physician, and Physician in Ordinary to H.M. the Queen, Windsor Castle.

1884 REID, THOMAS WHITEHEAD, Surgeon to the Kent and Canterbury Hospital; St. George's House, Canterbury, Kent.

Elected

1881 *Rice, George*, M.B., C.M., Sutton, Surrey.

1889 *Rivers, W. H. Rivers*, M.D., St. John's College, Cambridge.

*1871 *Roberts, David Lloyd*, M.D., Obstetric Physician to the Manchester Royal Infirmary; Physician to St. Mary's Hospital, and Lecturer on Clinical Obstetrics and Gynæcology at the Owens College, Manchester; 11, St. John street, Manchester.

1889 *Roberts, H. Leslie*, M.B., C.M., 46, Rodney street, Liverpool.

1873 *Robertson, William Henry*, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital; Buxton, Derbyshire.

*1888 *Robinson, Frederick William*, M.D., C.M., Huddersfield.

1889 *Robson, Arthur William Mayo*, Professor of Surgery, Yorkshire College; Senior Surgeon, Leeds General Infirmary; 7, Park square, Leeds. *Trans.* 4. *Pro.* 1.

1885 *Rockwood, William Gabriel*, M.D., Colombo, Ceylon.

1850 *Roper, George*, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; and to the Royal Infirmary for Children and Women, Waterloo Bridge road; Oulton Lodge, Aylsham, Norfolk. C. 1879-80.

1889 *Ross, Daniel McClure*, M.D., Cedar Lodge, Littledown Road, Bournemouth.

1863 *Rowe, Thomas Smith*, M.D., Consulting Surgeon to the Royal Sea-Bathing Infirmary; Union crescent, Margate, Kent.

1882 *Roy, Charles Smart*, M.D., F.R.S., Professor of Pathology in the University of Cambridge; Trinity College, Cambridge.

1871 *Rutherford, William*, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1891 *Ruffer, Marc Armand*, M.D., Medical School, Cairo.

Elected

1856 SALTER, S. JAMES A., M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

†*1855* SANDERSON, JOHN BURDON, M.D., LL.D., D.C.L. Durham, D.Sc., F.R.S., Regius Professor of Medicine in the University of Oxford; 64, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

1867 SANDFORD, FOLLIOTT JAMES, M.D., Surgeon-Major, Medical Officer of Health of the Drayton Union Rural Sanitary District; Surgeon to the Market Drayton Dispensary; and Consulting Physician to the Market Drayton Cottage Hospital; Market Drayton, Shropshire.

1886 SAUNDBY, ROBERT, M.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; Professor of Medicine, Mason College; 83A, Edmund street, Birmingham.

1891 SAUNDERS, FREDERICK WILLIAM, M.B., B.C., Chieveley House, near Newbury, Berks.

**1861* SCOTT, WILLIAM, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1887 SIDEBOTHAM, EDWARD JOHN, M.B., Erlesdene, Bowdon, Cheshire.

1857 SIORDET, JAMES LEWIS, M.D., Villa Labrolles, Mentone, Alpes Maritimes, France.

1886 SMITH, HOWARD LYON, Buckland House, Buckland Newton, Dorchester.

1885 SMITH, JAMES GREIG, M.B., C.M., Professor of Surgery, University College, Bristol; Surgeon to the Bristol Royal Infirmary; 16, Victoria square, Clifton, Bristol. *Trans.* 1.

Elected

1894 *Smith, Robert Shingleton*, M.D., Deepholm, Clifton Park, Clifton, Bristol.

1868 *Solly, Samuel Edwin*, Colorado Springs, Colorado, U.S.A.

1891 *Stevens, Cecil Robert*, M.B., B.S., Marwood House, Honiton, Devon.

1854 *Stevens, Henry*, M.D., late Inspector, Medical Department, Local Government Board, Whitehall; Durham Lodge, St. Margaret's road, Twickenham.

1884 *Stewart, Edward*, M.D., Brook House, East Grinstead.

†*1859* *Stewart, William Edward*, 68, Brunswick place, Brunswick square, Hove.

**1879* *Stirling, Edward Charles*, M.D., Senior Surgeon to the Adelaide Hospital; Lecturer on Physiology in the University of Adelaide, South Australia [care of Messrs. Elder and Co., 7, St. Helen's place].

1865 *Stokes, Sir William*, M.D., M.C., Surgeon to the Meath Hospital; 5, Merrion square north, Dublin. ***Trans.*** 1.

1871 *Strong, Henry John*, M.D., Consulting Surgeon to the Croydon General Hospital; Colonnade House, The Steyne, Worthing.

**1890* *Sympson, E. Mansel*, M.D., B.C., 3, James street, Lincoln.

1870 *Tait, Lawson*, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. ***Trans.*** 6.

1886 *Teale, Thomas Pridgin*, M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.

1890 *Thomas, William Robert*, M.D., Little Forest, Bath road, Bournemouth.

1891 *Thomson, John Roberts*, M.D., Monkchester, Bournemouth.

1883 *Thursfield, Thomas William*, M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.

Elected

1880 *Tivy, William James*, 8, Lansdowne place, Clifton, Bristol.

**1871* *Trend, Theophilus W.*, M.D., Physician to the Royal South Hants Infirmary; 1, Grosvenor square, Southampton.

**1881* *Treves, William Knight*, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 *Trotter, John William*, formerly Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1873 *Turner, George Brown*, M.D., The Lodge, Hemel Hempsted, Herts.

1881 *Tyson, William Joseph*, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne Gardens, Folkestone.

1854 *Waddington, Edward*, Hamilton, Auckland, New Zealand.

**1868* *Walker, Robert*, Clovelly, Bideford.

1887 *Wallace, Edward James*, M.D., Holmbush, Grove road, Southsea.

1867 *Wallis, George*, Consulting Surgeon to Addenbrooke's Hospital; 6, Hills road, Cambridge.

**1883* *Walters, James Hopkins*, Surgeon to the Royal Berkshire Hospital; 15, Friar street, Reading.

1894 *Ward-Humphreys, George Herbert*, Oriel Lodge, Cheltenham.

1846 *Ware, James Thomas*, Tilford House, near Farnham, Surrey.

1861 *Waters, A. T. Houghton*, M.D., Consulting Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.

1840 *Webb, William Woodham*, M.D., Ault-sur-Somme, Somme, France.

Elected

1874 *Wells, Harry*, M.D., San Ysidro, Buenos Ayres, S. America.

1882 *Wharry, Charles John*, M.D., 14, Ewell road, Surbiton, Surrey.

**1881* *Whitehead, Walter*, F.R.S. Ed., Senior Surgeon to the Manchester Royal Infirmary, Manchester and Salford Lock Hospital, and Manchester and Salford Skin Hospital; Professor of Clinical Surgery, Owens College, Victoria University; 499, Oxford road, Manchester. *Trans.* 1.

1885 *Whitla, William*, M.D., Professor of Materia Medica and Therapeutics, Queen's College, Belfast; Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.

1852 *Wiblin, John*, The Hermitage, Clewer, Windsor. *Trans.* 1.

**1870* *Wilkin, John F.*, M.D., M.C., The Warren, Beckenham, Kent.

1837 *Wilks, George Augustus Frederick*, M.D., Stanbury, Torquay.

**1883* *Willans, William Blundell*, Much Hadham, Herts.

**1859* *Williams, Charles*, Senior Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1887 *Wilson, Arthur Hervey*, M.D., 504, Broadway, Boston, U.S.A.

1863 *Wilson, Robert James*, 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1889 *Wise, A. Tucker*, M.D., Davos Platz, Switzerland.

**1850* *Wise, Robert Stanton*, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.

Elected

1879 *Woodward, G. P. M.*, M.D., Deputy Surgeon-General; 157, Liverpool street, Hyde Park, Sydney, New South Wales.

1892 *Wright, Almroth Edward*, M.D., Ch.B., Oakhurst, Netley, Hants.

ANNUAL MEETING.

Monday, March 2nd, 1896, at 5 p.m.

The President, on taking the chair, called upon Mr. R. W. Parker (Hon. Secretary) to read the minutes of the last Annual Meeting, and of the Special General Meeting held on June 11th, 1895. These were confirmed and signed.

The President nominated Dr. de Havilland Hall and Dr. Rolleston as Scrutineers, and requested them to superintend the Ballot. The President then declared the Ballot open till six o'clock, and called upon

Dr. Mitchell Bruce (Senior Hon. Secretary) to submit the following

Report of the Council.

The Council is pleased to report the continued prosperity and usefulness of the Society. The number of Fellows on the roll is 787. Twenty-three new Fellows have been admitted during the year, including ten Honorary Fellows who were nominated by the President and Council as being eminently distinguished in medicine, surgery or the allied sciences. Sixteen Fellows have died, including two Honorary Fellows of great distinc-

tion, namely, Mr. Huxley and M. Pasteur. Eight Fellows have resigned, and one has ceased to be a Fellow by reason of the non-payment of his annual contribution.

The papers accepted by the Society during the last twelve months show a considerable increase in number as compared with the previous year; and their value has been worthy of the high traditions of the Society. There is reason to believe that the increase in the number of communications is an evidence that the Fellows appreciate the rules which were made last March to provide for the earlier publication of papers.

The average attendance at the meetings has been 41 Fellows and 8 visitors, which shows the increased interest taken in the work of the Society. The standard of discussion has been well maintained.

In accordance with the resolution of the Council, confirmed at the last Annual General Meeting, two discussions on special subjects have been held during the year, after careful selection and arrangement by a Standing Committee of the Council elected for the purpose. The first discussion was opened by the President, and was on the subject of "Affections of the Nervous System occurring in the Early Stages of Syphilis;" the second was devoted to the question of the "Latency of Parasitic Germs and Specific Poisons in Animal Tissues." Both discussions were very successful: each occupied two evening meetings, and attracted many Fellows and visitors. The 'Proceedings' containing verbatim reports of these discussions were published as special numbers, and constitute valuable additions to the literature of the subjects.

On May 18th the Society was deprived of the services of one of its Treasurers by the death of Deputy Surgeon-General Bostock, C.B., who had been a Fellow since 1846, and had filled the important office of Treasurer for a period of seven years. Mr. Warrington Haward was elected to the vacant office at a Special General Meeting held on June 11th.

The Honorary Librarians report as follows:

"The Honorary Librarians are glad to be able to report that the Library has made very satisfactory progress during the past twelve months. As will be seen by the Treasurers' statement, a larger amount has been disbursed for books and binding than in any previous year; and the use of the Library has been commensurate with the increased expenditure.

"The arrangement under which new books are hired from Mr. Lewis continues to work well; and there is ample evidence that it gives great satisfaction to the Fellows, who are by this means enabled to peruse the most recently published books, which either would not be ordered at all, or which could not be procured until after the monthly meeting of the Council. During the year 154 volumes have been obtained from Mr. Lewis.

"The Library Committee has under consideration the question of providing a new Reference Catalogue upon a plan which will enable the entries to be kept up to date more readily than is possible with the Reference Catalogue now in use."

The following is the Report of the Committee on the Medical Climatology and Balneology of Great Britain and Ireland:

"The first volume of the Report of the Committee on the Climatology and Balneology of Great Britain and Ireland was published in July last by Messrs. Macmillan, and was presented at a meeting of the Society in October by the chairman, Dr. Ord.

"This volume deals with the climates of the south of England and the principal mineral springs of Great Britain.

"The Committee is at present engaged in the pre-

paration of a second instalment of the Report, which will deal with the climates of the remainder of England and Wales and with the minor mineral springs.

"The Committee has also under consideration the best means of dealing with the climates of Scotland and Ireland."

The foregoing bare official record should not be allowed to pass without a warm acknowledgment of the indebtedness of the Society to the contributors to this important work. In bulk alone it far exceeds any Report previously issued by the Society, and it is difficult to over-estimate its practical value to the profession and the public. It fills a much felt want in medical literature, and when completed will constitute a standard work of reference of great scientific importance.

The Committee appointed to investigate the subject of Suspended Animation in the Drowned has been actively pursuing its inquiry during the year. The difficulty of obtaining suitable subjects for experiment is very great, but the Committee hopes to be able soon to complete its investigations.

The Honorary Treasurers report as follows:

"The financial position of the Society remains satisfactory. The receipts of the Society during the year 1895 amounted to £4358 2*s*. 4*d*., and the expenditure to £4380 6*s*. 4*d*. The balance with which the year commenced was £933 2*s*. 8*d*., whilst the year 1896 commences with practically the same amount, namely, £910 18*s*. 8*d*.

"Among the receipts one item requires notice, the special receipt from Messrs. Duveen of £100, which was received in consideration of the Society undertaking not to interfere with their proposed alterations in Dering Yard.

"In the expenditure certain sums, namely, £8 for the address to the Nizam of Hyderabad, and £23 2*s*.. expenses connected with the meeting

of the British Medical Association in London, may be considered as accidental. The sums of £150 for highly remunerative alterations of premises, and £61 15*s*. 2*d*. for legal expenses, mainly in connection with granting leases to our tenants, may also be regarded as items not likely to recur in similar amounts.

"The Honorary Treasurers wish to draw the attention of the Council and the Society to the falling off in the number of entrance fees, which is the only unsatisfactory feature of the year's working. Taking the five preceding years, they find that the average number of Fellows joining the Society was thirty-one, and they cannot but regard it as a serious matter that during the year 1895 only ten new Fellows were introduced. Whilst the stability of the Society is assured from the value of its property, the Honorary Treasurers wish to bring prominently before the Society that its prosperity depends on the number of its Fellows."

The House Committee has held seven meetings during the past twelve months. In the course of the year this committee has undertaken the supervision of all bills (other than Library bills) not included in petty cash, so as to relieve the Council of a task which seemed more nearly connected with house-management than with the scientific work of the Society. The business of the Society has gone on very prosperously, the letting of the meeting-rooms having exceeded the estimate (£100) made of that item of income. In this as in other matters the Society is indebted to the unceasing watchfulness of the Resident Librarian.

Dr. CHURCH, the Senior Treasurer, submitted and explained the audited Statement of Accounts (see pp. xcii, xciii).

The PRESIDENT moved, and Dr. W. HOWSHIP DICKINSON seconded—"That the Report of the Council, together with the Treasurer's audited Statement of Accounts, be adopted and printed in the next volume of 'Transactions,'" and invited discussion thereon.

Dr. THEODORE WILLIAMS.—As we seem to have a large balance in hand this year, it occurs to me that it might be possible to apply part of it towards paying off the debentures. In other respects the report appears to be altogether satisfactory.

Dr. HARE.—As a former Treasurer, and to a certain extent responsible for the statement that for some time to come our repairs will probably not come to more than £100 a year, I wish to call attention to the item in this year's report which comprises a sum of £249 1*s.* 10*d.* for "repairs, alterations, &c." I should have been glad if it had been found possible to put under a separate heading the disbursements for repairs strictly so called as distinct from alterations. It would then be seen that a considerable amount of this sum was not really for repairs, but for what I may call reproductive alterations. Our Treasurer has mentioned that by the expenditure of £150 in alterations which could not be foreseen when the building was made, as the room altered was occupied by a charity from which we received a merely nominal rent, we have been able to obtain an increase of income of £100 a year. I congratulate the Society upon its very flourishing condition. Everything seems to be most satisfactory, and as Fellows we may sleep in peace knowing that our Society is prospering.

Dr. CHURCH.—Dr. Williams probably knows as well as I do that while we are under an obligation not to pay less than £100 a year towards paying off the debentures, we may pay as much more as the Council may direct. I may point out that this is really not a balance-sheet, but

a mere statement of actual receipts and actual payments within a given period, and for this reason it may give a more favorable idea of our position than the circumstances would justify. Out of the large balance of £910 we had to pay £660 on January 1st for interest on debentures.

The Report and accounts were unanimously adopted.

The PRESIDENT delivered the

ANNUAL ADDRESS (see p. xcvii).

On the motion of Mr. GEORGE POLLOCK, seconded by Dr. THEODORE WILLIAMS, resolved—"That the best thanks of the Society be given to the President for his valuable services during his term of office, and for his address just delivered."

On the motion of Dr. KENNETH MACLEOD, seconded by Mr. JOHN CROFT, resolved—"That the best thanks of the Society be given to the retiring Vice-President, Dr. C. J. Hare, for his valuable services during his term of office."

The PRESIDENT.—I can bear my testimony to the very great services rendered by Dr. Hare to the Society. Though it is not incumbent on Vice-Presidents to attend Council meetings, Dr. Hare has throughout been most regular in his attendances, and has given us the most valuable assistance.

Dr. HARE briefly acknowledged the vote.

On the motion of Mr. TIMOTHY HOLMES, seconded by Dr. GEORGE THIN, resolved—"That the best thanks of the Society be given to the retiring Honorary Secretary, Dr. Mitchell Bruce, for his zealous and valuable services during his term of office."

The PRESIDENT.—I am often credited with knowledge which I do not possess, but certainly no one knows more than myself how much work Dr. Bruce has done for the Society. During the last two years various modifications have been introduced into our procedure which involve heavy additional work for the Secretaries, and it

is mainly owing to his assiduity that things have proved so satisfactory.

Dr. Bruce.—I am very grateful for the kind remarks in appreciation of such services as I have been enabled to render the Society during my term of office, but during the two years of office my duties have brought me into contact with many fellows with whom I was not previously acquainted, with many of whom I have formed valuable friendships. Before sitting down I should like here publicly to thank our Resident Librarian, Mr. Mac Alister, for the valuable services he has rendered to the Society. In the matter of precedents which guide the Council he is never at a loss.

Dr. Althaus.—I have to propose a vote of thanks to the retiring members of Council for their assistance on a body which exercises such a great influence on the progress and welfare of the Society. This Council has carried out great reforms, and has put an end to the bad old system by which papers were read first and selected afterwards. Papers were refused admission to the 'Transactions' sometimes because they were too short or too long, and this to my knowledge has prevented authors from bringing forward interesting researches and investigations. I remember an occasion when the Society had to adjourn after the minutes had been read because there was nothing on the programme. This is a thing which under the new *regime* is not likely to happen again. I have therefore great pleasure in moving—

"That the best thanks of the Society be given to the retiring members of Council, Dr. Frederick Roberts, Dr. Frederick Taylor, Mr. Reginald Harrison, and Dr. Fitz-Patrick, for their services during their respective terms of office."

Dr. Gibbons seconded the Resolution, which was then unanimously agreed to.

The Scrutineers reported that the Officers and Council for the ensuing year had been duly elected as follows:

President.—William Howship Dickinson, M.D.

Vice-Presidents.—John Harley, M.D.; James Edward Pollock, M.D.; John Langton, Sir William MacCormac.

Honorary Treasurers.—William Selby Church, M.D.; J. Warrington Haward.

Honorary Secretaries.—Norman Moore, M.D.; Robert William Parker.

Honorary Librarians.—Samuel Jones Gee, M.D.; Rickman J. Godlee, M.S.

Members of Council.—John Abercrombie, M.D.; William Ewart, M.D.; David Ferrier, M.D.; Felix Semon, M.D.; Francis Charlewood Turner, M.D.; Arthur Edward J. Barker, Sir William B. Dalby, John Hammond Morgan, Edmund Owen, Robert Alexander Gibbons, M.D.

Having inducted the President elect into the chair and invested him with the President's badge of office, Mr. Hutchinson handed the new President the silver master-key, and said—This, sir, is the master-key of these premises. My handing it to you is no mere empty ceremony; it symbolises that you have the power and the right at any hour of night or day to visit and inspect all parts of this building. When your period of office expires I hope you may transfer this insignia of office to one whom you honour and esteem as highly as I do yourself.

The PRESIDENT (Dr. DICKINSON).—It does not become me on this occasion to do more than to express my gratitude for the honour you have done me in electing me to occupy this chair, an honour which I profoundly appreciate. I heartily thank my predecessor for the gracious way in which he has given effect to the formal resolution of the Society. In conclusion I may say that when my term of office expires it will be to me an almost unexpected gratification if I can look back on a period equal to the one through which he has passed.

Receipts

	£	s.	d.	£	s.	d.	£	s.	d.
Balance on 1st January, 1895:									
Cash in hand				99	19	10			
,, at Bankers				833	2	10			
							933	2	8
Subscriptions, Fees, &c.:									
429 Annual Subscriptions at £3 3*s.* ..	1351	7	0						
40 Non-resident Subscriptions at £1 1*s.*	42	0	0						
Composition Fees (Life) ..	26	5	0						
Entrance Fees	63	0	0						
				1482	12	0			
Transactions and *Proceedings:*									
Sold by Messrs. Longmans ..	69	11	4						
,, Mr. A. K. Lewis ..	1	3	3						
,, Resident Librarian ..	9	0	8						
				79	15	3			
Rents received				2683	2	9			
From Messrs. Duveen & Co.				100	0	0			
Interest:									
On Permanent Endowment Fund ..				12	12	4			
							4358	2	4
							£5291	5	0

MARSHALL HALL

The amount of Stock (Consols) standing to the credit of this

PERMANENT

New South Wales 4 % Inscribed Stock

W. S. CHURCH, M.D.,
J. WARRINGTON HAWARD, } *Treasurers.*

10*th February*, 1896.

Payments

	£	s.	d.	£	s.	d.
Rent, Rates, &c.				221	13	1
Lighting, Cleaning, and Heating				389	10	0
Repairs, Alterations, &c.				249	1	10
Meeting Expenses				29	6	8
Printing, Stationery, and Stamped Envelopes	120	15	3			
Stamps (other than above)	8	5	4			
				129	0	7
Officers and Servants:						
Salaries and Wages				800	0	4
Library:—Books and Binding				571	3	9
'*Transactions*' and '*Proceedings*'				290	3	10
Debentures, Interest on				1392	0	0
" *redeemed*				100	0	0
Auditors' Fee				10	10	0
Bank Charges and Cheques				5	13	4
Address to Nizam of Hyderabad				8	0	0
British Medical Association (reception)				23	2	0
Scientific Committees				46	18	11
Law Charges				61	15	2
Miscellaneous Payments				52	6	10
				4380	6	4
Balance:						
Cash in hand	218	11	4			
" at Bankers	692	7	4			
				910	18	8
				£5291	5	0

MEMORIAL PRIZE FUND

Fund on the 31st December, 1895, was ... £626 18 9

ENDOWMENT FUND

.. .. .. £326 7 3

Audited and approved.

WOODBURN KIRBY, MUNDY, & Co.,
Chartered Accountants,
19, Birchin Lane, E.C.

LIST OF PAPERS.

N.B.—The Council of the Royal Medical and Chirurgical Society deem it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, and opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its *Transactions*.

PAGE

I. On Posture in its Relation to Surgical Operations under Anæsthetics: by Frederic Hewitt, M.D.Cantab., Anæsthetist at the London Hospital, Charing Cross Hospital, and the Dental Hospital of London; and Marmaduke Sheild, M.B.Cantab., F.R.C.S.Eng., Assistant Surgeon to St. George's Hospital, and to the Royal Hospital for Children and Women 1

II. Renal Colic in Infants: by R. A. Gibbons, M.D., M.R.C.P., Physician to the Grosvenor Hospital for Women and Children 41

III. On Prevesical Abscess: by Ernst Michels, M.D.Berlin, F.R.C.S.Eng., Surgeon to the German Hospital 63

IV. Ecchymoses from Natural Causes: by H. A. Lediard, M.D., Surgeon to the Cumberland Infirmary . 75

V. The Exceptions to Colles' Law: by George Ogilvie, B.Sc., M.B.Edin., M.R.C.P.Lond., Physician to the Hospital for Epilepsy and Paralysis, Regent's Park 87

VI. The Parasite of Malaria, as observed in the Malarial Fevers of the South of Spain: by Robert J. Marshall, M.B., C.M., and George Thin, M.D. . 101

PAGE

VII. A Note on the Appearances found in the Tissues in a Fatal Case of Pernicious Malaria at Sierra Leone: by GEORGE THIN, M.D. 143

VIII. Excision of a Portion of Liver for Tumour: by MAYO ROBSON, F.R.C.S.Eng., Senior Surgeon to the General Infirmary, Leeds, and Professor of Surgery in the Yorkshire College of the Victoria University . 159

IX. A Case of Neoplasm Cyst of the Brain treated by Operation: by CHARLES A. BALLANCE, M.S., Surgeon to the National Hospital for the Paralysed and Epileptic, Queen Square; Assistant Surgeon to St. Thomas's Hospital, and to the Hospital for Sick Children, Great Ormond Street . . . 167

X. On the Effects produced by the Retention of Foreign Bodies for Lengthened Periods in the Bronchial Tubes: by RICKMAN JOHN GODLEE, M.S., Surgeon to University College Hospital, and to the Hospital for Consumption, Brompton, &c. 197

XI. Note on the Endemic Fever of the Mediterranean: by Surgeon-Captain MATTHEW LOUIS HUGHES, Army Medical Staff, Malta. Communicated by Dr. GEORGE THIN 209

XII. Observations and Experiments on Intestinal and Gastro-intestinal Anastomosis: by WALTER EDMUNDS, M.C., and CHARLES A. BALLANCE, M.S. . . 255

XIII. On the Use of Guaiacum in the Treatment of Chronic Gouty Affections, and its Value in warding off Acute Attacks: by Sir ALFRED B. GARROD, M.D., F.R.S. . 313

XIV. Case of Splenic Anæmia (spléno-mégalie primitive), with an account of the disease: by SAMUEL WEST, M.D. . 323

LIST OF PLATES

PLATE		TO FACE PAGE
I.	The Parasite of Malaria (ROBERT J. MARSHALL, M.B., C.M., and GEORGE THIN, M.D.)	142
II.	A Note on the Appearances found in the Tissues in a Fatal Case of Pernicious Malaria at Sierra Leone (GEORGE THIN, M.D.)	158
III.	Excision of a Portion of Liver for Tumour (MAYO ROBSON, F.R.C.S.)	166

ADDRESS

OF

JONATHAN HUTCHINSON, F.R.S.,

PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 2ND, 1896.

ANOTHER year having passed away, it is again my pleasant duty to pass in brief review the principal events of the Session. Twelve months ago I had to announce that the Council had decided upon certain not unimportant modifications in our procedure, both as regards our meetings and the publication of papers. These have now been fairly on their trial, and I trust it may be recorded that they have met with approval, and that they promise well for the increasing success of our Society in the future. During the past year all papers accepted for reading have been *ipso facto* accepted for publication. Authors of papers no longer encounter any risk of the humiliation of having a paper, which has been read, subsequently declined as unsuitable for publication. All that the Council now reserves to itself is the power of determining whether such publication shall be in the 'Transactions' or 'Proceedings.' Every paper that has been deemed worthy to be read is now, in one or the other form, published in full. It is believed that this modification of procedure is working well, and our secretaries have had

during the current session a larger number of papers offered than has been the case in any similar period for some years past.

The more prompt publication of papers which has been effected by dividing the year's volume into three parts has also, it is believed, been an acceptable change, and one which by the satisfaction given to our Fellows generally has more than compensated the Society's officials for the additional trouble which it involves. A collateral advantage of much value has been that we have been able to produce, some little time in advance of every meeting, the full text of the paper about to be read. This has, I feel sure, added much to the interest and importance of our discussions.

In accordance with another of the recommendations of your last year's Council, a certain number of our meetings have been devoted to Debates upon special topics. These Debates have been arranged by your Council through a standing Committee which has been responsible for the selection of subjects. So far as the attendance of Fellows can be held to be a criterion, these Debates have been very successful, for it has been more than double the average of our ordinary meetings. I trust also that it may be said that each one of our three Debates has been of a character to mark a not unimportant epoch in our knowledge of the subject concerned.

The aim of the first of these was to elicit evidence as to the nature of an important and large group of those affections of the nervous system which occur in connection with syphilis, and to prove that they really belong in all essential characters to the early and secondary group. It had of course long been noticed, and by many observers, that the affections referred to did in reality often occur during the first year after infection, but it had been too much the custom to assume that when they did so, they were examples of irregular and precocious development of the malady. Little or no attempt had been made to differentiate them from those

of the later periods which really belong to the tertiary stage, —that of gummata and degenerative changes. The outcome of our discussion was, if I mistake not, to accept many of the affections referred to as belonging definitely to the secondary or humoral period; being as much a part of it as are the general skin eruptions and the affections of the throat and eyes. It was shown that in most of them the primary pathological changes were not in the nerve-elements themselves, nor in the bones or fibrous structures, but rather in the peri-vascular spaces, and the tissues of the smaller blood-vessels. Nor is this distinction one merely of names or of classification. It includes our correct conception of the process involved, and upon it must be based both our treatment and prognosis.

To call the snowdrop and crocus autumn flowers, and to admit at the same time that they do really bloom in early spring, would not be more inconsistent than to continue to name as tertiary those phenomena of syphilis which occur in its early stages.

I cannot close my reference to this discussion without gratefully remarking that it was the means of eliciting from Dr. Gowers and Dr. Ferrier amongst many others very able contributions to our knowledge of the subject.

The second Debate took very wide ground, possibly too wide. It dealt with no less a subject than the possibilities as regards the latency of specific or parasitic germs. The recent developments of our knowledge in reference to these germs have been very large, and it cannot be a matter of surprise that modern discoveries as to bacteria and their allies should have brought into great prominence the doctrines of contagion. These doctrines, emphasised as they appeared to be by the discovery of some specialised form of parasite in most of the more peculiar types of the inflammatory process, were rapidly monopolising the attention of the younger and more energetic race of pathologists. Constitutional predispositions and other more general influences were being pushed aside, and the implantation from without of

a vital contagium was accepted with but little hesitation as the true and solely efficient cause of a majority of our diseases. The Debate arranged by your Council's committee did not go the length of challenging these conclusions, nor did it deal in any way with the very important question of congenital or acquired constitutional predispositions as giving proclivity to parasitic attacks. That topic may possibly be taken up on a future occasion. It had been tacitly assumed in most instances of bacterial or cryptogamic infection that the implantation of the parasite occurs very shortly before the manifestation of its effects, and it was to this assumption that the consideration of the Society was requested. The questions asked were as to the possibility of germs,—like the seeds of plants in unpropitious soil or too deeply buried, —retaining vitality during long periods, and manifesting activity, after such a long duration of absolute quietness that the date of their introduction might be lost in doubt. Might it even be possible that such specific germs should have been acquired by a parent and transmitted as such to the offspring? In seeking answers to these questions it seemed needful to take facts from very different sources, and hence the association in the Debate of such diverse maladies as hydrophobia, syphilis, tuberculosis, leprosy, and even ringworm. From the first of these we learn more conclusively, perhaps, than from any other malady, the startling fact that the local process of incubation of a specific virus may be delayed for almost indefinite periods, whilst from leprosy we gain evidence of an almost equally unexpected kind as to the length of the period which may elapse between the introduction of the specific cause of a constitutional malady and the earliest outward manifestations of its presence.

In this Debate the general bacteriological facts were most ably expounded to us by Dr. Washbourn, who contributed much that was novel and original, and gave support from experimental investigations to the belief

that these specific germs might under favorable circumstances retain their vitality for almost indefinite periods of time. Dr. Fowler, who undertook the exposition of the subject in reference to Tuberculosis, upheld the same view, and was, if I mistake not, prepared to admit the possible transmission of the tubercle bacillus from parent to child. The facts as regards Leprosy, which are to some extent cognate with those as to tuberculosis, were ably stated to us by Dr. Abraham, and those in reference to Syphilis by Mr. James Ernest Lane. On the latter subject, as might perhaps have been expected, both the introducer and those who spoke after him found great difficulty in distinguishing between the true latency of specific germs and the long delayed manifestation of their more remote results. What is called "latent syphilis" by no means always implies latency of specific germs.

It might perhaps be noted as a defect in this Debate that it was too much one-sided, for no one attempted in any material degree to invalidate the arguments of those who upheld the doctrines of almost unlimited latency. Without venturing to allege that this Debate brought forward much that was really new, it may yet be asserted that it attracted attention to a very important phase of pathological inquiry, and that the subjects with which it dealt are certain in the future to receive more adequate recognition.

The third and last Debate of the session is but just over. In contrast with those which preceded it, it has dealt with a single topic, that of newly discovered parasites of malarial fevers. Had circumstances then been favorable this subject would have been chosen as that for the first Debate of the session. It was obvious that the topic was one which ought to be brought before such a Society as ours, and concerning which opportunity should be offered to our Fellows to bring forward their individual experience, and give the profession the benefit of their criticisms. It is precisely for such subjects as this

that the new arrangement of special Debates is, I believe, designed. We have less reason to regret the delay of six months than we might have felt, since it has secured for us the able paper by Dr. Marshall and Dr. Thin, containing original observations in a new field.

I must not refer in any detail to the various papers which have been read before us further than just to express the opinion that they have been in interest and value well up to the high standard which the Society proposes for its contributions. One of them, the paper by Dr. Hewitt and Mr. Marmaduke Sheild, excited so much interest that it was necessary to prolong the discussion over two evenings.

I have next, gentlemen, to undertake the duty, in some respects a melancholy one, of recalling to your memory the names, and I trust somewhat more than the names, of those of our Fellows who have, during the past year, fallen from our ranks. The list is a long one, and it is perhaps almost unexampled in respect to the professional eminence of those whom it comprises. We may note, however, with a sad satisfaction that those who have fallen were, almost without exception, men well advanced in life, and that we have not to mourn as in some former years the premature loss to our profession of those who had seemed destined to take a high place in its ranks. It is sad enough to lose a veteran leader and an old friend, but it is yet more so, if we take a full view of the facts, to see a young and ardent spirit, with his life's work still before him, cut down by one of the many perils which surround professional life, and consigned "to sleep without his fame." There are not in the list which is before me more than one or two concerning whom any feeling of this kind can be entertained.

It may not be without its interest if, in passing, I state that taking ten names out of my list,—and they are those of the most distinguished and include some of the hardest workers,—the average age at death was no less than seventy-four years.

For reasons stated last year I shall attempt nothing in the form of biographical memoirs, but must content myself with brief allusions to some of the principal facts.

Of *Dr. Launcelot Andrews*, who died at the early age of thirty-one, I believe it may be recorded that he was a type of all that is excellent in the department of practice which he had chosen.

Mr. Hugh Lane, of Bath, was, like Dr. Andrews, engaged in family practice, and like him enjoyed the esteem of all who knew him. He was at the time of his death, which was after a very short illness, on the staff of the Bath United Hospitals.

Amongst those whose deaths most nearly touch this Society we have the name of *John Ashton Bostock*. Although Mr. Bostock was eighty years of age he was still in sound health and a regular attender of our Council meetings, holding as he did up to the time of his death the office of Treasurer. He was one whose judgment was much valued by all who knew him. He was the son of the foremost pathological chemist of his day, and he had himself gained many honours in connection with the Army Medical Service. He was at the time of his death C.B. and Deputy-Surgeon-General.

One of the names which have to be removed from our list of Honorary Fellows is that of *Baron Larrey*. He was the worthy son of Napoleon's great military surgeon, and had himself seen some service in the field. He was a member of the French Institute and of the Academy of Medicine, and was highly esteemed, both in his native land and abroad, for his surgical attainments and his high personal character. Baron Larrey was in his eighty-seventh year at the time of his death. He had been almost to the last a regular attendant at the meetings of the Academy of Medicine.

In *Sir Thomas Longmore* and *Sir Thomas Crawford* our public services have lost two who had been their chief ornaments. The one was Honorary Physician, and the other Honorary Surgeon to Her Majesty, and both were

honoured by many titles and decorations which had been won in professional pursuits. Both had been engaged in the Army Medical School at Netley, where Sir Thomas Longmore was for long the Professor of Surgery. Sir Thomas Crawford had risen to the position of Director-General of the Army Medical Service (resigned in 1889). The one was near his eightieth year at the time of his death, and the other had reached that of seventy-two; the one was a native of London, the other of the north of Ireland. Two more energetic and able servants of the public it would be difficult to mention. Of Sir Thomas Longmore it may be justly said that his teaching, based as it was upon enormous personal experience, had a very large share in the progress made during the last half century in all that relates to Military Surgery.

It may, perhaps, to some seem strange if I link together two men in many respects so different as *Mr. James Dixon* and *Sir William Savory*. Yet they were men of not dissimilar character, and they entertained a strong regard for each other. The latter once told me that he had read through Mr. Dixon's book on Diseases of the Eye, "not, you know, that I care for the subject, but it is such a treat to find a medical work written in good English." Both had cultivated style, and had attained a high degree of finish. Both were metropolitan, not only by birth and education, but by deeply-rooted preference. Both were men of the most upright and straightforward character, and both were instinctively and intensely conservative, although neither, I believe, ever took any part in the politics of the day. I have heard a tradition, however, that Mr. Dixon's grandfather had been a leader of the Tory party in the City of London.

Mr. Dixon was fifteen years Mr. Savory's senior. He was Assistant Surgeon to St. Thomas's Hospital at the time when Mr. South and Mr. John Henry Green were its surgeons. He was eighty-two years of age at

the time of his death, whereas Sir William Savory was only sixty-seven.

Mr. James Dixon was a well read man of highly cultivated tastes. He was a good draughtsman and a skilful operator. For a time it seemed likely that he would take the lead in ophthalmic practice in London. The late Mr. George Critchett was, I believe, elected at the same time upon the staff of the Moorfields Hospital. Mr. Dixon, who subsequently had great influence there, was, I have been told, one of those who exerted their interest to secure for the institution the services of one whose genius destined him to become a powerful rival. Mr. William Bowman, although Assistant Surgeon to King's College Hospital, was at that time chiefly known as a physiologist, and had done little or nothing in reference to Diseases of the Eye. With characteristic energy and ability he threw himself into the new work, and his fitness for the vocation soon became manifest to all. Both he and Mr. Dixon soon resigned their appointments to their other hospitals, and devoted themselves wholly to the more attractive speciality. It was a period of progress. The ophthalmoscope was introduced, iridectomy was invented, and new methods for the extraction of cataract were devised.

In reference to all these Mr. Dixon's inherited conservatism placed him at a disadvantage. He could not easily move with the times, and all novelties encountered his distrust. I do not think that he ever did any operation for the extraction of cataract excepting the old one with Beer's knife and without anæsthetic. This he could do most dexterously, and with great success. It would not surprise me if it were true that he never did an iridectomy for glaucoma. It was my privilege to be for many years his assistant at Moorfields, and it was upon his patients that my earliest observations as to interstitial keratitis, choroiditis, and notched teeth were made. I owed much to his guidance. He was one of the first to be convinced that the conditions just mentioned were really due to

inherited syphilis, and in his article in 'Holmes' System of Surgery' he proposed that the term "interstitial keratitis" should be replaced by that of "syphilitic keratitis."

Mr. Dixon was at one time very influential in the Council of this Society. He had held the offices of Vice-President, Treasurer, and Librarian, and our 'Transactions' contain three papers by him. He retired rather early in life from all active duties in connection with the profession, and for the last twenty had lived in seclusion at his country home near Dorking. In this retreat, however, he did not wholly neglect the profession which he had adorned, and the readers of our weekly journals encountered with pleasure, every now and then, short scholarly letters from his pen. These were usually in elucidation of some question in medical history or philology.

The influence which *Sir William Savory* exerted over medical education was great, and much of it will be lasting. He was an advocate for an additional year of compulsory study long before that measure was—at the instance, I believe, of the Medical Council—finally adopted. Although he was not the proposer of an additional examination, yet it was undoubtedly by his advocacy that this suggestion took effect. His opinions on these and other subjects were like the rest of his character—definite, emphatic, and sound. He insisted upon the paramount importance of thorough knowledge of anatomy and physiology as the basis upon which that of surgery must be built, and he did much, both in connection with the medical school with which he was associated and with the examining bodies, to improve the methods of teaching these subjects. It may be doubted, however, whether his sympathies with the modern methods of clinical investigation were equally strong. Although himself a sound practical surgeon, it is possible that he was not really much enamoured of clinical work. All forms of specialism he contemned, and no modern surgeon more absolutely escaped

the association of his name with any particular class of diseases than he did. He almost wholly ignored the usefulness of special hospitals as means to clinical training, and I feel sure that I do not misrepresent his opinions in saying that he held that the more closely a student was induced to restrict himself to the lecture rooms and hospital wards of his own school, the better for his education. In these matters he was resolutely metropolitan, and he regarded the opportunities offered by smaller institutions, or by private practice, as comparatively worthless. There was a clearness and consistency in this view which was characteristic of his modes of thought. That he was an opponent of all specialism in examinations need not be said. With the design of encouraging higher education by inducing many to seek the higher qualification, he was an unflinching upholder of the privileges of the Fellows of the Royal College of Surgeons. Of the Members he held that they ought to be content with their diploma, and grateful for the opportunities afforded them for acquiring further knowledge in the College library and at the College lectures. That they had any claim to share in the government of the College he stoutly and successfully denied. How great was the force of his individual authority in this matter may be estimated by the fact that within a year of his death the Fellows themselves have passed a resolution by a large majority agreeing voluntarily to abandon most of the privileges in dispute.

Sir William Savory was, we all remember, a man of commanding presence and great personal influence. His opinions were always expressed concisely, clearly, and strongly; no one could mistake his meaning, and what he said always carried weight. As it was remarked of Buffon, that if not Nature's Historian he was at any rate her Orator, so it might be asserted of him. His address at the Cork meeting of the British Medical Association, his Hunterian Oration, and other later performances, proclaimed him the Orator of Surgery. They were finished compositions, delivered with ease and fluency, and they

commanded the rapt attention of crowded audiences. It is a common mode of praise to say of a man of wide general ability that he would have risen to eminence in any one of the professions. Of no one has it ever been possible to assert this with more emphatic truth than of Sir William Savory. Either in the Church or at the Bar he would certainly have come into the foremost rank, indeed it is possible that the bent of his mind was more definitely towards one of these than it was to that of practical surgery. His superlative ability as a leader was recognised in his election in three successive years as President of the College of Surgeons, the office not having been previously held by any one for more than a single year.

Savory was not a voluminous writer, but all that he did write was well done, and concerned important subjects. Our 'Transactions' contain several papers from his pen, and it is of interest to remark that his original researches on the possibility of the passage of poisons from the mother to fœtus have supplied important facts in reference to two of the subjects which have been under our discussion during the present session: I refer to the latency of germs and the scope of Colles' law.

Although Sir William had attained the age of sixty-seven, yet his career of usefulness was by no means at an end. He had but just concluded his duties as one of the Royal Commission on the subject of a new University for London, and he still took a warm interest in promoting the realisation of the recommendations which it had given. During the last six years he had been one of the most active members of the Royal Commission on Vaccination, and I may be permitted, on the part of his colleagues, to bear personal testimony to their deep sense of loss which his death has caused in their counsels.

Mr. Arthur Edward Durham was born at Northampton in 1833, and was sixty-two years of age at the time of his death. For some years he had been much out of health, though by no means disabled from work. At the College of Surgeons Mr. Durham had held the office of Vice-

President. The disabling influence of a physical defect prevented his ever becoming an examiner or a candidate for the presidentship.

Of Arthur Durham it may be recorded that the qualities of his heart to some extent overshadowed, or to speak more appropriately, overshone those of his mind. Every one thought and spoke first of his lovable nature, of his goodness, and his unselfish devotion to others. Thus, although we all knew it, there was some danger that we might forget to remark that he was also a well-trained anatomist and a brilliant scientific surgeon. Mr. Durham's experimental researches on the state of the brain during sleep were carried out as long ago as 1860. By these he gained his first reputation as an original investigator. He subsequently wrote many valuable papers in the 'Guy's Hospital Reports,' in 'Quain's Dictionary of Medicine,' in 'Holmes's System of Surgery,' and in the 'Transactions' of our own Society. He had been for thirty-three years on the Surgical Staff of Guy's Hospital, and was accounted a bold, skilful, and prudent operator as well as one whose diagnosis was hardly ever at fault.

By the death of *Sir George Buchanan*, at the age of sixty-five, the important department of Sanitation and State Medicine has lost its leader. In saying this I do not forget that his eminent predecessor, Sir John Simon, is still with us, for he is in the enjoyment of well-earned and honourable rest, whereas Buchanan was still to the front in active work. The labours of the Royal Commission on Tuberculosis, of which during the latter part of its sittings Buchanan was the Chairman, had but just concluded when his much-to-be-regretted death took place. It may be suspected, indeed, that the devotion to duty which, although he was aware that he was the subject of incurable disease, led him to continue at his post in this Commission, had its influence in conducing to the fatal event.

Buchanan, in common with five others of the distinguished men whom I have had to mention to-day, was a

Londoner by birth. He was the son of a family practitioner residing in Myddelton Square, and was educated throughout at University College and its School. Having in the early part of his career been associated with the Fever Hospital and that for Sick Children, he soon found for himself a special vocation in connection with Public Health. He became Health Officer of the district of St. Giles's and rose rapidly until in 1880 he was appointed Principal Medical Officer to the Local Government Board. This post he held for twelve years, and I cannot better sum up his labours than by quoting from one of the obituary notices which appeared soon after his death.

"His admirable introductory reports, dealing with the highest branches of sanitary science, in the seventeen volumes of Reports of the Medical Officer, which were issued from his department during his term of office, were valuable and suggestive contributions to the general bulk of knowledge. Whether we have regard to his work in reference to vaccination as a branch of preventive medicine; to hospital accommodation in relation to disease repression; to his studies of the ætiology, pathology, and bacteriology of infectious diseases, alike of human and animal origin; to his care for the sanitary advance of the country; or to his well-directed labours for the safeguarding of our shores from exotic disease;—indeed, from whatever view-point we regard his unceasing endeavours to maintain England's proud position as the leader of the nations in matters sanitary, we are bound to think of him as foremost amongst sanitary workers."

On his retirement from his Government appointment Buchanan received the distinction of knighthood, one never more meritoriously bestowed. From his medical brethren he received the yet higher honour of a subscription which enabled him to endow a Triennial Gold Medal, to be granted by the Royal Society for distinguished services in sanitary science.

Of *Thomas Henry Huxley*, who was at the time of his death one of the Honorary Fellows of this

Society, it may be said with truth that no man more definitely lived into the very structure of the century than he. Although he took no part in the government of his country, and although even in the growth of science we do not affix his name to any great discovery, yet it is true that no one more influentially guided the development of modern thought. The opinions of England, of Europe, of America, of all civilised parts of the world, are to-day largely what Huxley made them. Vast and varied as were his scientific attainments, it is yet in the vocation of teacher that we place him in the highest rank. Endowed with a marvellous mastery of facts, clear and logical in his modes of thought, fearlessly devout in his love of truth, he easily took precedence of all others as the exponent of progressive thought during a period which will not improbably be regarded in the future as one of the most important epochs in the world's history. We have not necessarily become agnostics, yet the creed of most men of thought is now to a large extent that which Huxley taught us, and the opinions which a quarter of a century ago he had to defend, are now the commonplaces of general knowledge. To his genius, his industry, his unflagging zeal, and his resolute determination to see things as they are, his victories were due.

I will make no attempt to recapitulate Huxley's original work, or to give the titles of the almost innumerable essays and papers in which his researches are enshrined. It must be sufficient now to remind you that his position was acknowledged by the highest honour which British science can give—the Presidency of the Royal Society; whilst his services to the cause of national education were recognised in his becoming one of Her Majesty's Privy Council.

Upon a small house in the Rue des Tanneurs, in Dôle, may be read an inscription in letters of gold which tells us that *Louis Pasteur* was there born on December 27th, 1822. It is recorded that from his earliest childhood he displayed very remarkable intelligence, so that it is

said that his father, who was a working tanner, often expressed a confident ambition that he might one day become a professor. That ambition was fulfilled, and the tanner's son became in succession a professor of many sciences, and he finally died the most renowned discoverer of his age. Pasteur's life has been sketched by so many, and its main facts have been so recently revived in our memories, that it is not needful that I should attempt to enumerate them. I excuse myself the more willingly because the master pens of Paget and Lister have already discharged this labour of love in a manner which I cannot hope to emulate. It must suffice to say that, inspired by a passion for truth and endowed richly with every faculty which can ennoble the human mind, M. Pasteur devoted a long life to the service of humanity in the pursuit of experimental knowledge.

M. Pasteur was, as has been well said, "chemist, microscopist, and naturalist in one." Although, perhaps, his chief fame is in reference to chemistry, yet he may be rightly named the father of bacteriological science. Not amongst the least of what he accomplished we must place the fact that his were the researches which inspired the mind of Lister, and thus led to results which can scarcely be regarded as of less importance than those obtained by the master himself.

I do not think that you will blame me if I venture to quote to you the touching phrases in which Sir Joseph Lister, who enjoyed Pasteur's personal friendship, has described his character.

"I must content myself with a few words regarding his personal characteristics. His acuteness as an investigator in seizing upon essential points, and his wonderful lucidity of judgment, were only equalled by the patience with which he pursued what he termed *la méthode expérimentale;* and his enthusiasm was always tempered by dispassionate caution. In doing battle with the fallacious doctrine of spontaneous generation he was a keen controversialist, but his utterances were always charac-

terised by transparent truthfulness. His rare modesty and entire freedom from affectation made intercourse with him easy and delightful. Anyone who reads the account he gave in the *Comptes Rendus* of the case of the little boy on whom he first ventured to employ anti-rabic injections in the human subject will see clear indications of another feature of his character—loving tenderness of heart. His splendid early work in physics and chemistry proved, indeed, how dearly he loved pure science for its own sake; yet it was undoubtedly the great joy of his later researches that they directly promoted the good of mankind. In Pasteur the world has lost a personality as beautiful as it was great."

With Pasteur's great name, and Lister's eloquent tribute to his memory, I end my list, for time compels me to leave wholly aside two or three whom I would otherwise gladly have mentioned.

Now, gentlemen, I must conclude. In transferring as I do to-day the office of your President, to which you elected me two years ago, I can but express a deep sense of many short-comings. I have, however, done my best, and I now have to thank you heartily for the manner in which you have on all occasions so kindly seconded my efforts. To the members of the Council, and in a most especial manner to our Secretaries, Dr. Bruce and Mr. Parker, the expression of my warmest appreciation of their valuable assistance is due.

Let me just add that the honour of having held the Chair of the Royal Medical and Chirurgical Society is one which, while memory lasts, I shall never forget.

ON

POSTURE IN ITS RELATION TO SURGICAL OPERATIONS UNDER ANÆSTHETICS

BY

FREDERIC HEWITT, M.A., M.D.CANTAB.,
ANÆSTHETIST AT THE LONDON HOSPITAL, CHARING CROSS HOSPITAL, AND THE DENTAL HOSPITAL OF LONDON;

AND

A. MARMADUKE SHEILD, M.B.CANTAB., F.R.C.S.ENG.,
ASSISTANT SURGEON TO ST. GEORGE'S HOSPITAL, AND TO THE ROYAL HOSPITAL FOR CHILDREN AND WOMEN

Received August 5th—Read October 22nd, 1895

INTRODUCTION

THE posture of a patient prior to, during, and after a surgical operation under a general anæsthetic is a subject of considerable importance, not only to the surgeon and anæsthetist, but in many cases also to the patient himself. It is, moreover, a subject of some complexity, owing to the numerous circumstances which must be taken into consideration before any definite statement can be made as to this or that particular posture. Possibly this complexity may help to explain the absence from our literature of any systematic and exhaustive monograph on

posture. But be this as it may, the time appears to us to be opportune for opening up the subject and directing attention to certain conclusions at which, after carefully arranging the facts at our disposal, we have been able to arrive. A moment's reflection will show that the questions at issue are by no means so simple as we could desire. We have to consider the various postures which the body and head may be required to assume during surgical operations, and these are exceedingly numerous. We have to inquire what influences these postures are capable of producing upon the respiration and circulation, not only of subjects in a good or fair state of health, but in those with serious affections of the vascular, respiratory, or nervous system. And in dealing with postures during general anæsthesia we have to take into account the kind of anæsthetic employed, its method of administration, and the depth of anæsthesia at the time to which our remarks apply.

The importance of the subject to the surgeon is greater than might at first sight appear, for in deciding upon this or that posture for a surgical operation something more than mere operative convenience ought to be taken into account. It would, of course, be irrational to expect a surgeon to operate upon a patient placed in such a position that the operation would in consequence become a matter of serious difficulty. But if the adoption of a particular posture would prejudicially affect the patient, and if some other posture, although not quite so convenient to the surgeon, would be unattended by such objections, there can be no doubt that the surgeon should, in the interests of his patient, accept the compromise, and place the patient in the safer though perhaps less convenient position. But the subject of posture appeals to the surgeon through other channels; for the position chosen may very materially influence the condition of the parts upon which the operation is being performed. This may come about in one of three ways :—
(1) The posture may be such that, by favouring the

occurrence of jerky and exaggerated breathing, coughing, retching, or vomiting, it greatly interferes with that quietude which is essential to the surgeon ; (2) it may, by impeding or interfering with the respiration, lead to congestion, venous oozing, and inconvenient hæmorrhage ; or (3) it may, in response to the influence of gravity, advantageously or disadvantageously modify the blood-supply to the site of operation.

To the anæsthetist the subject is of equal if not of greater importance. With him the responsibility undoubtedly rests of maintaining or endeavouring to maintain a perfectly free air-way and unimpeded respiratory action from the commencement of his administration until the patient has acquired sufficient consciousness to be left unwatched. If the posture be one in which the trunk weight tells unfavourably upon free lung expansion, or one in which blood, vomited matter, pus, or other adventitious substances tend to gravitate into the laryngeal aperture, serious difficulties and accidents will be liable to arise. In addition to these considerations, recent physiological and clinical observations have shown that in the more or less vertical posture of the body, and especially under certain anæsthetics and in certain degrees of anæsthesia, the effects of gravity upon the circulation may be such as to cause threatening or fatal symptoms.

Lastly, the posture immediately before and after an anæsthetic is given is to the patient himself a matter of importance, not only because some postures are extremely uncomfortable when inhaling anæsthetics, and are really quite unnecessary, but because recovery from the effects of these agents takes place more readily and quickly in some postures than in others. Unpleasant after effects, also, may to some extent be avoided if the posture during the administration has been one in which mucus and saliva have drained away from the fauces, and have therefore not entered the stomach to be subsequently ejected.

It is hardly necessary to draw attention to the fact that a careful consideration of posture is particularly necessary in very obese subjects, in those whose respiration is from any cause embarrassed before or likely to become embarrassed during anæsthesia, and also in patients whose circulation, from functional or organic heart affections, emotional disturbances, loss of blood, shock, or any allied condition, is liable to be quickly influenced by gravity when the body is moved.

Speaking generally, it is far better for the patient to be rendered insensible on the table, bed, couch, or chair which has been provided for the operation than for him to be anæsthetised elsewhere and subsequently moved. The administration is easier, and insensibility is therefore more speedily and successfully produced; anæsthesia is less likely to be disturbed by the vomiting or retching which may be originated by moving a partly anæsthetised patient from one place to another; should struggling or undesirable symptoms arise during administration of the anæsthetic, they can be more easily encountered and managed if the patient be in a convenient position than if, for example, he be lying in the middle of a four-post bed; and, lastly, the patient is not anæsthetised for a needless length of time.

The preceding remarks are merely introductory, and we now propose to consider in detail various attitudes and positions adopted in surgical procedures, pointing out, as far as practicable, the various advantages, disadvantages, or actual dangers attending each. Though many classifications of so large a subject are possible, we think that the matter will be made clear by a consideration of it under the following headings:

I. The posture of the head in its relation to that of the trunk.

II. The influence of the force of gravity upon the circulation and respiration.

III. The postures of surgery individually considered:

(*a*) the dorsal, supine, or horizontal posture; (*b*) the dorso-lateral posture; (*c*) the lateral posture; (*d*) the latero-prone and prone postures; (*e*) the semi-recumbent posture; (*f*) the sitting posture; (*g*) special postures, viz. (1) the lithotomy, (2) the semi-inverted (Trendelenburg's), and (3) the knee-elbow and knee-chest postures.

IV. The posture of the patient after the operation.

I. The Posture of the Head in its Relation to that of the Trunk.

Observations extending over several years have convinced us that a faulty position of the head either before, during, or after the use of an anæsthetic may and occasionally does lead to serious difficulty. A patient is sometimes said to have "taken the anæsthetic badly," when the fault has lain not with the patient but with the surgeon or anæsthetist. Not only may difficulties, such as repeated swallowing, coughing, laryngeal spasm, general movement, cyanosis, venous turgescence, and the production of large quantities of mucus, arise from faulty postures of the head, but complications of an asphyxial character may readily supervene from this cause. It is therefore important that the circumstances which may contribute to these events should be understood.

(*a*) *Extension of the head and neck upon the trunk.*—Before an anæsthetic is administered, *i. e.* whilst the patient is conscious, extension of the head for more than a few seconds is attended by considerable discomfort, partly because the position is an unnatural and constrained one, and partly because swallowing becomes difficult or impossible, so that if an inclination to swallow should arise, that inclination cannot be gratified. Anyone, by throwing the head well back, and trying to swallow, may prove this for himself. It is not uncommon, especially in dental surgical practice, for a patient to sit or to be placed, as a matter of supposed necessity, in this very uncomfortable posture during the adminis-

FIG. 1.—Sitting posture, with head extended. Faulty.

tration of the anæsthetic (see Fig. 1). The administration is then very likely indeed to prove unsatisfactory, simply on account of the faulty posture of the head. If the nature of the operation be such that the head must be thrown back, the body should be thrown back also, *i. e.* into the semi-recumbent posture, so that the head is not extended. Extension of the head is equally uncomfortable, and, as a rule, equally unnecessary, when the patient is placed in the supine posture immediately before the administration of an anæsthetic.

In partially established or moderately deep anæsthesia —such an anæsthesia, for example, as that often regarded as advisable in some mouth and nose operations—head extension is very liable to be attended by difficulties, no matter whether the patient be sitting, semi-recumbent, or supine. It is hence advisable, if this extension be desired, as may be the case in some operations upon the neck, to secure deep anæsthesia. Sometimes, when the head is extended and the anæsthesia is not deep, the presence of blood, mucus, saliva, &c., at the superior

aperture of the now unprotected larynx may not only readily excite retching, coughing, and futile deglutition movements, but the patient's general muscular system may share in the spasmodic efforts to close the larynx, or to expel what has entered it. Usually all that is needed in such cases is to put the patient more deeply under the anæsthetic, and the extension of the head will no longer cause the difficulties referred to. This matter is of such importance that we should like to bring it into prominence by an illustration. Let us suppose that a powerfully built man, of alcoholic habits, rather bloated in appearance, with thick neck and well-developed jaw muscles, is about to have a trivial operation performed, such as the removal of a tooth and the opening up of the antrum for drainage. The operation is not regarded as of sufficient gravity to warrant the patient going to bed, or to call for the use of an operating table. He is accordingly placed upon a couch, in the semi-recumbent posture, with his face in the mid-line and his head ex-

FIG. 2.—Semi-recumbent posture, with head extended. Faulty.

tended over the sloping end of the couch (see Fig. 2). Now this posture is one of the worst that can be chosen; and whatever anæsthetic be employed, and whatever method be adopted, difficulties will be certain to be encountered under the circumstances here narrated. The anæsthetic will be "taken badly"; the patient will be

almost certain to struggle and to become opisthotonic; and unless very skilled assistants be at hand, delay will be experienced in introducing sufficient quantities of the anæsthetic to keep such a patient deeply under its influence. Moreover, the larnyx being comparatively unprotected by reason of deglutition movements having been rendered difficult or impossible, the anæsthetic vapour, mucus, pus, and possibly blood will be liable to excite innumerable reflex troubles. It is in such cases as these that one of two things is liable to happen: either the operation has to be abandoned because the patient cannot be properly anæsthetised, or he becomes so asphyxiated that the medical attendant vows he will never give him chloroform again. All that is needed in such a case is to anæsthetise the patient whilst he is lying in a comfortable posture (*i. e.* either upon his side or upon his back, with the head turned to one side), to secure a very deep anæsthesia before the operation is begun, to keep the patient well under the anæsthetic till the end of the operation, and to see that his head remains turned to one side throughout to facilitate drainage. Some surgeons operate for adenoid growths, cleft palate, and other conditions, with the head very completely extended over the

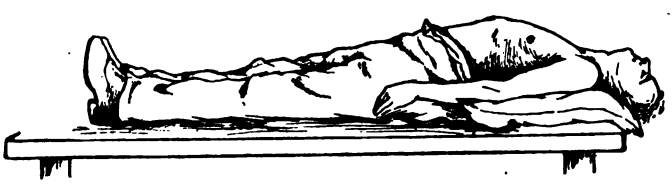


FIG. 3.—Dorsal posture, with head completely extended over end of table.

end of the table (Fig. 3); but if this be done the patient should be previously thoroughly anæsthetised, otherwise difficult breathing, often erroneously attributed to the presence of large quantities of blood, will be liable to arise for the reasons already given. The nasal passages, moreover, must be free, otherwise breathing may not proceed.

Extension of the head has different effects from those just narrated when the patient is thoroughly anæsthetised; for the larynx is now incapable of being excited by irritants. It must not be forgotten, however, that the larynx is all the more prone to be quietly invaded by blood or vomited matter, supposing that the posture is such that these substances would gravitate towards its orifice. A little reflection will show that there are three principal postures in which the gravitation of fluids towards the larynx is most likely to arise, namely the dorsal, the semi-recumbent, and the sitting, always supposing that the face is in the mid-line. The entrance of blood into the larynx in considerable quantities is liable to occur when there are several conditions present at the same time, these conditions being—

(1) A deep anæsthesia;

(2) The dorsal, semi-recumbent, or sitting posture; and

(3) The face in the mid-line and the head extended.

When all three factors are present, and hæmorrhage is free, the entrance of large quantities of blood into the larynx is inevitable unless repeated sponging be practised. If either factor is absent, the chances are greatly lessened, or perhaps entirely removed. Thus if the anæsthesia be light, and the laryngeal and pharyngeal reflexes intact, coughing and swallowing, if the latter can possibly take place, may prevent the invasion. Again, if the patient be in any other posture than those mentioned, or be in the dorsal posture with the head on the side, blood will escape from the mouth. In the semi-recumbent, and more especially in the sitting posture, this turning of the head to one side does not have the effect of facilitating the escape of blood, so that the chances of this fluid entering the larynx become greatly augmented. This helps to explain, in our judgment, the difficulties and fatalities which have been recorded in connection with mouth and nose operations in these postures; and we would strongly urge their abandonment in such procedures, except under certain

circumstances, to which we shall allude when dealing specially with the sitting and semi-recumbent positions. Lastly, if the third factor—head and neck extension—be absent, the larynx retains its natural protector, the epiglottis; and although embarrassed breathing from the presence of blood may still arise, yet there is not that risk of blood entering in such quantities as when the laryngeal aperture is unprotected.

(*b*) *Flexion of the head and neck.*—As pointed out by Bowles,[1] this has the effect of lessening the calibre of the air-way, and of favouring stertor and respiratory embarrassment. If the patient be supine, the use of several pillows under the head (and not under the shoulders) is open to objection; for when insensibility is established, asphyxial symptoms from the cause just mentioned will be liable to complicate the anæsthesia. This is especially observed in patients with short thick necks, and in those whose air-passages are naturally narrowed by such causes as glandular swellings, adenoid growths or enlarged tonsils. Recovery from the effect of the anæsthetic will similarly be retarded if such a posture be permitted.

(*c*) *Rotation of the head upon the trunk.*—This has little or no influence upon respiration, so that when the patient is in the supine posture the head should invariably be kept turned to one or other side throughout the administration, provided that such a posture is convenient to the operator. Lives have more than once been lost by want of attention to this simple precaution. If the patient be lying in the dorsal posture with the face turned upwards, difficulties in the administration will be more liable to arise than if the face be kept to one side: mucus and saliva will tend to induce cough, and will, when swallowed, favour retching and vomiting; the apparently abnormal tendency towards return of consciousness on the part of the patient leads to the use of a large quantity of the anæsthetic; and disturbances

[1] 'On Stertor, Apoplexy, and the Management of the Apoplectic State,' 1891.

both of respiration and of circulation attend or follow the inconvenient symptoms displayed by the patient. It is highly probable that the comparatively large number of deaths which have taken place in ophthalmic operations under anæsthetics may be partly explained on these grounds. If the head be turned well to the side (see Fig. 5) mucus and saliva tend to flow out of the corner of the mouth, so that coughing, swallowing, retching, and vomiting are far less likely to arise than if the face be in the mid-line. Moreover, if a loose tooth should become dislodged, or hæmoptysis or epistaxsis arise, difficulties which might otherwise occur will be avoided. But the greatest argument, perhaps, for this particular posture is, that should vomiting take place, as is often the case, for example, in cases of intestinal obstruction, the vomited matter will be ejected from the mouth, and will not be sucked into the larynx, as has so often happened, with fatal consequences.

II. The Influence of the Force of Gravity upon the Circulation and Respiration.

The influence of the force of gravity upon the circulation has long been recognised, although no precise and systematic research upon the subject has, until quite recently, been accomplished. The observations of Snow will be referred to when dealing with the sitting posture. The Hyderabad Chloroform Commission found that the blood-pressure in the carotid arteries of lower animals was considerably modified by posture, in obedience to the force of gravity.[1] When the hind feet were lowered to the floor, so as to place the animal in the vertical position, a considerable fall of blood-pressure in the carotid artery occurred; when the animal was placed horizontally the pressure was restored. Inversion of the body had exactly the opposite effect to elevation, the blood-pressure in the

[1] See Report, p. 28.

carotid rising, but falling again to its previous state when the animal was placed horizontally. The most complete work on the effect of gravity upon the circulation is that of Dr. Leonard Hill.[1] In this paper the author gives the results of his investigations on the "Influence of the Force of Gravity on the Circulation." Dr. Hill made a series of observations upon the normal intra-cranial tension in a patient of Dr. Clay Shaw's who had been trephined. Similar experiments were also carried out upon animals, and similar results obtained from all. The animals were anæsthetised and placed upon a board, with the limbs fully extended in the longitudinal axis of the body. The results obtained by this physiologist are too numerous to mention in detail, but some of the more important may be here enumerated:

1. The force of gravity must be regarded as a cardinal factor in dealing with the circulation of the blood.

2. The splanchnic vaso-motor mechanism has the important duty of compensating for the simple hydrostatic effects of gravity in changes of position.

3. When the compensatory power of the splanchnics is damaged by paralysis induced by severe operations, injuries to the cord, asphyxia, or some poisons, as chloroform or curare, then the influence of gravity becomes of vital moment.

4. In the feet-down position, when the compensatory power of the splanchnics is destroyed, the blood drains into the abdominal veins, the heart empties, and the cerebral circulation ceases.

5. The feet-up position at once restores the action of the heart and the cerebral circulation, and firmly bandaging the abdomen has the same effect.

6. So long as the heart remains normal the blood-pressure cannot fall if mechanical pressure be applied to the abdominal veins.

7. If the heart be affected, as by chloroform or curare, the restoration of pressure is incomplete, and the

[1] 'Proc. Royal Society,' vol. lvii, No. 342.

heart may be stopped by the inrush of a large quantity of blood, caused by too rapid an application of pressure on the abdomen. More work would be thrown upon the heart than in its impoverished condition it could perform.

From Dr. Hill's researches it would, therefore, appear that chloroform can, by destroying the compensatory mechanism, rapidly kill the animal if it be placed with the abdomen on a lower level than the heart. He advises elevation and compression of the abdomen in chloroform collapse. Ether, on the other hand, only slowly paralyses the compensatory vaso-motor mechanism, and this when given in enormous doses.

Dr. Hill also finds that in animals the feet-down position inhibits respiration, and the feet-up position accelerates it, and that these results are abolished by dividing the vagi. He believes, therefore, that they depend upon the stimulation of sensory nerve-endings by changes of tension, brought about by the alterations of position. He also observed that in the feet-down posture the respiration was thoracic in type, and the abdomen was retracted; whilst in the feet-up posture the respiration was diaphragmatic, and the abdomen was freely expanded. Dr. Hill concludes by pointing out that these types of respiration tend to compensate for the effects of gravity on the circulation. The retraction of the abdomen in the feet-down position must tend to support the abdominal veins, whilst the free thoracic inspirations aspirate blood towards the heart. In the feet-up position, the full and free expansion of the abdomen withdraws all obstacles to the compensatory dilatation of the abdominal veins. The practical application of these researches is obvious. In chloroform syncope, in severe shock, or dangerous hæmorrhage, the feet-up position should be assumed, and the abdomen should be bandaged. The results of this physiologist, so far as the effects of chloroform upon the circulation are concerned, will be seen to agree largely with those of Macwilliam, and to be opposed to those of the Hyderabad Commission.

III. The Postures of Surgery individually considered.

(*a*) *The dorsal, supine, or horizontal posture.*—The purely dorsal posture is not, perhaps, the best for the induction of anæsthesia. Most persons pass into natural sleep either in the dorso-lateral or lateral, not in the purely dorsal position. The customary dorsal posture

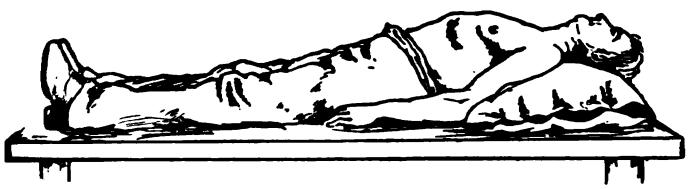


Fig. 4.—Customary dorsal posture. Head slightly extended. Face turned upwards. Faulty.

with the head slightly extended (Fig. 4) is certainly open to objection for reasons already given. As will be subsequently mentioned, the dorso-lateral posture is usually the best in which to commence the administration of an anæsthetic. If the patient prefers to lie horizontally, the head should be turned to one or other side.

When the patient has been placed under the anæsthetic we may say that, with the exception of special cases, the dorsal posture is the best, provided that the head is neither flexed nor extended, and that it is kept

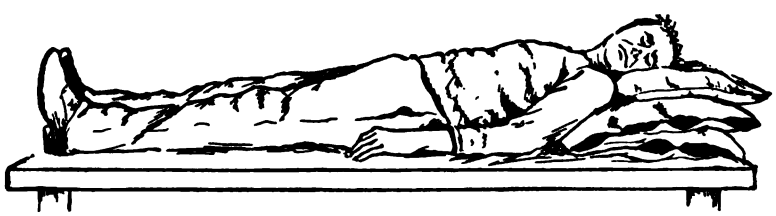


Fig. 5.—Dorsal posture. Face turned to one side. Correct.

turned to one or other side (Fig. 5). The main argument in favour of this posture as the most appropriate in ordinary surgical cases is that it is the one least likely to

prejudicially influence the respiration and circulation of an anæsthetised patient, always supposing that no abnormalities in the respiratory functions are present. In the absence of such abnormalities, chest expansion will take place more freely in this than in all other postures, save perhaps the semi-recumbent and the sitting. But if the respiration be principally or wholly diaphragmatic, as is often the case in very stout subjects, the dorsal posture may not be the most satisfactory, owing to the abdominal viscera having to be raised with each contraction of the diaphragm. It is an interesting fact that during general anæsthesia respiration is often to a great extent diaphragmatic, even in healthy subjects, the chest wall and sternum actually receding during the inspiratory phase, and rising slightly during expiration. Under such circumstances as these it is clear that the diaphragm would have more work to do than if the patient were lying partly or completely upon his side. But when the respiration is chiefly thoracic, and the chest expands with inspiration, the dorsal posture would certainly be preferable to the lateral. So far as the modifying effects of gravity upon the circulation are concerned, the dorsal posture is obviously the best.

A few words must here be said concerning mouth and nose operations upon anæsthetised patients lying in the dorsal posture. Should this position of the body be required, the head should, if possible, be kept turned to one side to facilitate the escape of blood. Should it be necessary, for the convenience of the operator, that the head should not be turned to one side, it should either be fully extended, in which case a deep anæsthesia is permissible and, as has been pointed out, advantageous, or it should be allowed to retain its natural relations to the trunk, under which circumstances repeated sponging with large coarse sponges will be needed, and the anæsthesia should only be moderately deep, in order that coughing and swallowing may persist. Considerable skill, experience, and judgment are needed for keeping up

anæsthesia during such an operation, for example, as that of removing adenoid growths from the naso-pharynx, when the patient is lying in the dorsal posture with the face turned upwards and the head not extended. In conducting such cases to a successful termination everything depends upon careful sponging and upon keeping up a particular degree of anæsthesia.

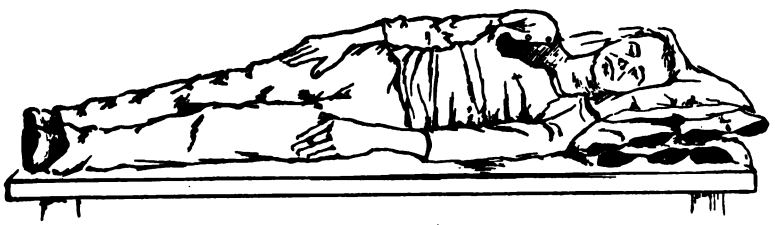


FIG. 6.—Dorso-lateral posture.

(*b*) *The dorso-lateral posture.*—This posture (Fig. 6) is a particularly appropriate one during the initial stages of the administration of an anæsthetic. It is more comfortable to the patient than the purely dorsal, and is less likely to interfere with thoracic expansion than the purely lateral posture. When everything has been prepared in the room in which the operation is to take place, and the patient is ready to be anæsthetised, he should be encouraged to adopt this posture on the operating table or bed, unless special circumstances are present rendering such a posture unadvisable or impossible. It is a simple matter, when once anæsthesia has been produced, to turn the patient either into the usual dorsal or into the lateral position. The dorso-lateral posture is, however, an excellent one for a large number of operations, being equally as good as the purely dorsal, and in many cases more convenient from the surgeon's point of view,—as, for example, in operations upon the breast, axilla, neck, face, mouth, nose, &c. It is, moreover, a very useful posture when respiration is wholly diaphragmatic.

(*c*) *The lateral posture.*—The lateral or side posture is one which is deserving of a wider application in surgery

than that which it at present enjoys. In this position, as was forcibly and clearly pointed out by Dr. R. L. Bowles[1] as far back as 1857, the air-way is less likely to be obstructed than in the dorsal posture, because the tongue tends to gravitate towards the dependent cheek. As the Fellows of the Royal Medical and Chirurgical Society are aware, Dr. Bowles has very successfully treated the coma of cerebral hæmorrhage by placing patients upon the side, a procedure which has the effect of stopping stertor, facilitating the escape of mucus, saliva, or vomited matters, and keeping one lung free from congestion, and therefore able to carry on respiration satisfactorily. The lateral posture is hence an excellent one, as a general rule, for patients recovering from the effects of anæsthetics (*vide infra*, IV).

Although it is clear that in the lateral posture full expansion of the subjacent half of the thorax cannot take place, this does not seem to constitute any great objection to the position, at all events in the great majority of cases, in actual practice. We have already drawn attention to the fact that respiration during anæsthesia is often to a great extent diaphragmatic, which in itself is sufficient to explain the usual absence of any respiratory embarrassment when patients are placed upon the side. Indeed, in very obese subjects, and in those with a distended abdominal cavity from ascites or other causes, breathing will be found to take place better upon the side than in the dorsal posture, owing to the fact that in the former posture the work of the diaphragm is considerably lessened.

Patients are often said to be lying in the lateral posture when this is not really the case. The hip and shoulder of one side should both be in contact with the operating table or bed, and the knees should be flexed. The mistake is often made of turning the shoulders but not the hips, and the body then gradually tends to return to the dorsal posture.

[1] Op. cit.

When the patient is lying wholly upon the side one cheek naturally rests upon the pillow, and in this posture many operations upon the jaw, mouth, pharynx, and nose may most advantageously be performed without any fear of embarrassed breathing from the blood which escapes into the upper air-passages. When it is anticipated that the hæmorrhage will be free, the head should be brought well to the edge of the table, and the face turned towards the floor (Fig. 7)—a position in which drainage takes place

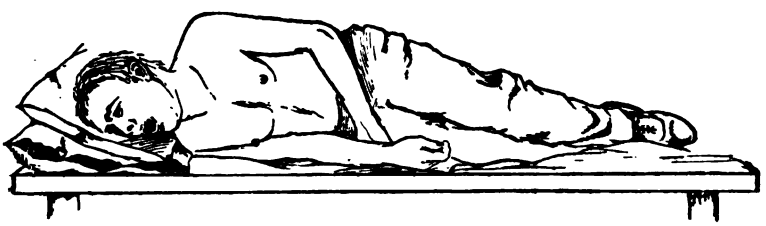


FIG. 7.—Lateral posture. Head brought to edge of table. Face turned towards floor.

very readily. The lateral posture is an excellent one for such operations as the removal of the upper jaw, the whole or a portion of the tongue, naso-pharyngeal polypi, adenoid growths, nasal polypi, &c. Some operators, recognising the advantages of having the head upon the side in these cases, attempt to secure this end by turning the head whilst the patient's trunk is in the dorsal posture; but the result is not so satisfactory, owing to the tendency of the head, in the moderately deep anæsthesia usually advisable in such cases, to regain its natural relations to the trunk.

The lateral posture being one that is often advisable in operations for empyema, it will be convenient to make some remarks here upon the posture of empyema patients in general. After a very large experience, Dr. Goodhart informs us that a patient with pus in the pleural cavity may assume for choice any position, the special intra-thoracic conditions of the case prompting the adoption of

that posture in which respiration can be carried on with the least possible exertion or distress. Concomitant pulmonary or bronchial disease may thus explain apparently unusual postures. Before proceeding further, a word may be said as to moving these patients from the bed to an operating table. Although this may be attended by some inconvenience, it is certainly the best plan in most cases. The patient should, however, be moved with great care and caution. He should not be permitted to raise himself, but should be carried *horizontally*. Then comes the question as to his posture during the induction of anæsthesia. Speaking generally, empyema patients should be permitted to assume that position which is most comfortable to them; and the surgeon, even though he may be inconvenienced, should endeavour to operate without changing the posture to any considerable extent. There can be no doubt that when nature dictates the best posture in a particular case, any marked deviation from that posture should, if possible, be avoided. The most rational plan certainly appears to be to administer the anæsthetic to the patient lying in the most favourable position, and then, should the exigencies of the operation be such that this must be modified or changed, to effect the change subsequently, vigilantly watching the effect. Should the patient choose to lie on his non-affected side, he will fortunately be in a convenient posture for the operation. This, however, is not common. Supposing that he experiences no greater discomfort in one posture than in another, he should be placed ready for the operation, so that when sufficiently anæsthetised no delay need arise. But should he experience discomfort or distress when an attempt is made to place him in the posture most convenient for the operation, some compromise should be effected, otherwise alarming symptoms may ensue. There are some cases in which the patient lies on his affected side, unwilling or unable to move, and in these the operator's convenience must give way to other considerations. The anæsthesia should only be partial.

the patient's trunk should be drawn over slightly beyond the edge of the table or bed, and the surgeon, sitting upon a low chair, will then be able to operate either without altering the posture of the patient or without disturbing him to any undesirable degree.

To make this most important matter clearer, let us suppose a patient to be lying on his right side, and the right pleural cavity to be full of pus. Dyspnœa being present, the patient should be gently lifted on to a high table, the affected side still being kept dependent. If the surgeon wishes to operate in the anterior axillary region, the thorax and abdomen of the patient must face the operator. Should the posterior axillary region be selected, the back of the patient faces the surgeon. But in either case it is essential that the thorax projects over the edge of the table, the patient being carefully held by assistants, with his shoulders and hips upon the table, and the affected side of the thorax projecting over its edge. When orthopnœa is present, it is better to operate without altering the patient's position than to lower the body.

Whenever the operation is such that the lateral or latero-prone posture will be required (as, for example, for operations upon the kidney, colotomy, &c.) the patient should be anæsthetised in the lateral posture, so that little or no subsequent alteration in position will be needed.

We have recently met with a minor complication attendant upon the lateral or latero-prone posture which deserves passing notice. We refer to pressure upon the brachial nerves. The musculo-spiral especially may suffer, and wrist-drop, with weakness of the extensors, arise. The causation of these cases is pressure, and they are analogous to those of wrist-drop seen in persons who have gone to sleep with the arm hanging over a chair. So far as we know, the symptoms are temporary, but they are apt to cause inconvenience and alarm to a nervous patient. In the lateral and latero-prone postures,

therefore, the position of the arm should be studied, and pillows arranged to take off pressure.

(*d*) *The latero-prone and prone postures.*—The experience of recent years has somewhat altered prevalent views as to these postures. The Chloroform Committee of the Royal Medical and Chirurgical Society of 1864 state in their Report,[1] "The prone position is inconvenient to the administrator, but entails no extra danger." With this statement we cannot, however, agree; and, as the subject is one of great importance it is well that the fallacies of former teaching should be recognised, and that teaching abandoned. The late Dr. C. E. Sheppard, in an able article on "The Administration of Ether in Operations requiring the Lateral or the Prone Position,"[2] deals *in extenso* with this subject; and no one can deny the correctness of his views. He quotes the observations of Dastre, who found that whilst it required the weight of 75 kilogrammes applied to the sternum to stop the respiration of a dog of 12 to 13 kilogrammes not under an anæsthetic, a weight of 58 kilogrammes would effect this when insensibility was produced, and that after one hour of chloroform anæsthesia a weight of 25 kilogrammes was all that was needed to arrest breathing. He further refers to certain experiments made by the Hyderabad Chloroform Commission, in which the animals rapidly succumbed under chloroform when their chests were encased in plaster-of-Paris jackets and the respiratory movements restricted. Dr. Sheppard then points out that in the semi-prone and prone postures (Fig. 8) the trunk weight must necessarily prevent free thoracic and abdominal expansion, and that, especially when anæsthesia is deep and respiration is in any way hampered, very undesirable symptoms of an asphyxial character may supervene. In his own practice he met with two cases in which, under chloroform, breathing came to a standstill as the direct result of the semi-prone posture, respiration

[1] 'Med.-Chir. Trans.,' 1864, p. 353.

[2] 'Brit. Med. Journ.,' July 11th, 1891.

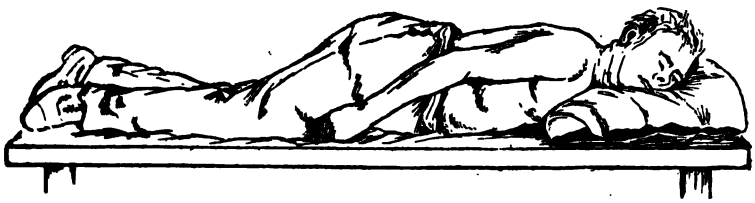


FIG. 8.—Latero-prone posture.

again starting when the patients were placed in the dorsal position. Dr. Sheppard speaks strongly in favour of ether as an anæsthetic in cases requiring the prone or semi-prone posture. Clinical experience seems to point to the fact that respiratory difficulty is most marked when the patient is placed in such a position that the weight of the body falls on the anterior axillary line and in front of it. The weight of the superincumbent arm must not be overlooked when the limb is bulky and the chest walls weak. In the latero-prone posture, therefore, the uppermost arm and shoulder should be kept raised from the trunk by means of a pillow or some appropriate contrivance.

From the above considerations it follows that fat, bloated persons, whose abdominal muscles are flabby, and whose chest walls are weak, can only be anæsthetised in the prone or latero-prone posture with some risk. This point should be borne in mind in connection with prolonged operations upon the kidney, and in the performance of laminectomy for spinal tumour or disease.

Marion Sims's posture, which is often employed in gynæcological practice for such operations as dilatation of the cervix, curetting, vesico-vaginal fistula, &c., is ordinarily termed the left lateral, but a more accurate definition would be the left latero-prone position; for the right knee is drawn upwards until the patient can be tipped over on the left breast ('American Text-book of Gynæcology'). It is plain that in this posture the left chest cannot freely expand, and the right must be embarrassed by the weight of the superincumbent upper

limb. The diaphragm, moreover, might be prevented from acting freely, owing to pressure upon the abdominal contents. In a very stout woman, and in a very long operation, the Marion Sims posture may, therefore, prove embarrassing to respiration.

The purely prone posture is rarely if ever really necessary, the latero-prone being nearly if not quite as convenient to the operator, whilst it is certainly preferable from the anæsthetist's point of view. Should the operator wish, however, that his patient should lie almost or completely prone, the anæsthetist may with advantage place a couple of small but firm pillows under the shoulders in order to facilitate thoracic expansion; or the pelvis may be similarly raised with the object of rendering abdominal movements easier of performance. We have found that by adopting this plan respiration has undergone distinct improvement.

(*e*) *The semi-recumbent posture.*—By the semi-recumbent posture we mean that in which the patient's body is about halfway between the vertical and the supine positions (Fig. 9).

FIG. 9.—Semi-recumbent posture. Head neither extended nor flexed. Correct.

It is a posture which is often adopted, to suit the convenience of the surgeon, for operations about the mouth

and throat,—as, for example, in the removal of tonsils and the extraction of teeth. We regard it, however, as an unsatisfactory one, except under certain circumstances, for the following reasons:—(1) Should the patient struggle or become rigid, the struggling and rigidity are more difficult to restrain than if he be lying horizontally upon his back, or sitting nearly vertically in a chair. (2) The tongue tends to gravitate towards the pharyngeal wall, and thus to favour stertor. (3) Mucus, saliva, vomited matters, and blood cannot so readily be made to flow from the mouth as in the dorsal and sitting postures. In the dorsal posture this object is readily achieved by keeping the head turned to one side, or by occasionally turning the head and shoulders to one side; in the sitting posture it is attained by tilting the whole body forwards from time to time. In the semi-recumbent posture, however, such procedures are difficult of execution, from which it follows that repeated swallowing movements, coughing, retching, and obstructed breathing are very liable to arise during mouth and nose operations upon a semi-recumbent patient. (4) *Chloroform* cannot be given with safety to patients in this posture (see II).

We have already alluded to the importance, as a general rule, of avoiding head and neck extension; and the remarks which we have made forcibly apply to the posture now under consideration. Whenever the semi-recumbent position is needed, the head should be kept as far as possible in a line with the trunk. Indeed, if this be done, difficulties will be far less liable to arise than if the head be permitted to become extended. The hypothetical case to which we referred when dealing with head and neck extension will fully illustrate our meaning.

Although the semi-recumbent posture is not a good one in most cases, it is nevertheless very suitable in a few special instances. For example, the removal of tonsils under general anæsthetics is more difficult, especially in children, than is generally supposed; but if the patient, who has been placed under ether, be propped up in the

semi-recumbent posture in front of a window, a better chance of successfully performing the operation will be afforded the surgeon than in any other position. Immediately after the removal of the second tonsil the patient should be lowered into the latero-prone posture, or tilted well forwards till the head is over the knees (see Fig. 11). In either of these postures all blood will flow out of the mouth, and will in no way embarrass breathing. Moreover, should it be necessary to remove any adenoid growths which may be present, this can be readily effected in one or other of these latter postures. Any brief nose or mouth operation may be similarly performed with the patient in the semi-recumbent posture; but longer operations upon these parts, for reasons which will be sufficiently clear from what has already been said, are not so satisfactorily accomplished as in other postures.

The semi-recumbent posture is to be preferred to others, at all events for inducing anæsthesia, when it is the only posture in which the patient can breathe with comfort. Thus it is not unfrequently adopted by patients suffering from extreme obesity, pulmonary affections, cardiac diseases, great abdominal distension from tumour or fluid, intra-thoracic aneurysm or tumour, and other conditions attended by dyspnœa. If such patients have to be anæsthetised at all, the anæsthetic should be given, with every care and precaution, in that posture which is most comfortable and in which respiration seems most freely to proceed. It is often possible, in many cases of this class, to substitute the dorsal for the semi-recumbent posture when once unconsciousness has been produced. This change, however, must be cautiously effected. In the case of extreme abdominal distension, capable of being relieved by operation, the body should be lowered as respiration improves by reason of the reduction in size of the abdomen.

We cannot conclude our remarks upon the semi-recumbent posture without venturing to hope that such a posture will, for the future, be abandoned in grave

operations upon the mouth and nose. A case recorded in the 'British Medical Journal' for February 25th, 1882, well illustrates the dangers of this posture. The patient, who was under ether, died during the removal of the upper jaw, the fatal symptoms being attributable to the entrance of blood into the trachea and bronchi, a complication largely dependent upon the semi-recumbent posture in which the operation was performed. We cannot too strongly deprecate the practice of performing these grave operations when the patient is in the semi-recumbent or sitting posture, more especially when experience teaches that the lateral position, although a little less convenient for the surgeon, renders the administration of the anæsthetic perfectly simple, and practically abolishes all risk from the entrance of blood into the air-passages.

(*f*) *The sitting posture.*—In the sitting posture the body of the patient is more vertical than in the semi-

FIG. 10.—Sitting posture. Head neither extended nor flexed. Correct.

recumbent position (Fig. 10). The trunk may be quite vertical if desired, though this is usually not necessary,

a very slight inclination backwards being more comfortable and convenient. The remarks which we have already made in a previous part of paper, on the posture of the head in relation to that of the trunk, apply in no small measure to the sitting posture. The combination of this posture with more or less extension of the head and neck is very liable indeed to give trouble, more especially in operations upon the mouth and nose.

Most of what we have said regarding the semi-recumbent posture will apply to the posture now under consideration. The sitting position is, however, preferable in most respects to the semi-recumbent. The only disadvantage which it possesses as compared to the latter is that the more vertical position of the trunk is favourable to the occurrence of syncope. This point will be specially referred to below. The sitting is a somewhat better posture than the semi-recumbent, because, should excitement or muscular rigidity arise, they can be more easily kept in check. Moreover the posture is a preferable one for most mouth and nose operations, because it is extremely easy from time to time to tilt the whole body forward in order to empty the mouth of blood or vomited matter. Thus, in long dental operations, when the patient has to be kept under ether for a considerable time in order that numerous difficult teeth may be removed, the ether inhaler, preferably one of Ormsby's pattern, may be kept applied to the face with the body tilted forwards for the escape of the blood (see Fig. 11). In this way there is not the objection to re-applying the ether that there undoubtedly is when the oral cavity contains blood, and the body is thrown back. This bent-forward posture is similarly advantageous during the removal of adenoid growths from the naso-pharynx by means of the natural or artificial nail. When a proper degree of ether anæsthesia has been secured, a mouth gag is inserted, the patient's body and head are bent forwards (Fig. 11), the operation is proceeded with, and all blood and detached vegetations escape freely from the mouth. The sitting

FIG. 11.—Bent-forwards posture.

posture has another advantage over the semi-recumbent, namely, that during the induction or maintenance of anæsthesia the tongue is less likely to gravitate towards the pharyngeal wall and to obstruct breathing.

Opinions have for many years been divided as to whether anæsthetics should be given to patients in the sitting posture. The point is one of great importance. The truth appears to be that it is safe to give certain anæsthetics by certain methods to certain patients whilst they are thus placed; whereas with other anæsthetics, other methods, and other patients the risk may be considerable.

Snow[1] held that there was no objection to the sitting posture when it was most convenient to the operator, provided that should faintness occur, the usual plan of placing the patient in the horizontal posture was adopted. He seems, however, to have had some slight misgiving in expressing this view, as he also states that he considers the horizontal posture the best for the patient. He administered chloroform to 949 patients in the sitting posture without any serious result.

[1] 'Chloroform and other Anæsthetics,' pp. 75 and 76.

The Chloroform Committee of the Royal Medical and Chirurgical Society state,[1] with regard to the administration of this anæsthetic, "In the erect or sitting posture there is danger from syncope." As we have stated above, the Hyderabad Chloroform Commission found that very considerable variations in the blood-pressure of animals were to be brought about in response to alterations from the horizontal to the vertical posture and *vice versâ*; but with regard to the possible combined effects of a surgical operation and the vertical posture, this Commission state,[2] "Various operations were performed on animals in the vertical position, but in no case was anything resembling dangerous shock produced."

Coming to more recent work still, we have fully referred to Hill's researches, from which we learn the important part played by the splanchnic vaso-motor mechanism in compensating for the simple hydrostatic effects of gravity in changes of posture—effects which have been long known to the profession. Dr. Hill has shown that it is damage to the compensating power of the splanchnics which is to be feared—a damage which may be brought about by conditions which are not infrequently at work in surgical operations under anæsthetics, *e. g.* severe operations, injury to the cord, asphyxia, the use of excesssve quantities of chloroform, &c.

Speaking in general terms, we may say that with nitrous oxide and with ether there appears to be no objection, so far as the anæsthetic itself is concerned, to the sitting posture. We need hardly say, however, that in the case of an exceedingly feeble circulation being present from any cause, either before, during, or after the administration, the sitting posture should not be permitted for an instant, but the patient should be placed horizontally. With regard to chloroform the case appears to be different, and there is good ground for the rule that

[1] 'Transactions' of the Society, 1864.

[2] 'Report of the Hyderabad Chloroform Commission,' p. 28.

during the use of chloroform patients should be lying down.

(*g*) *Special postures.*—Special positions are sometimes adopted by surgeons for certain operations, the principal of these being as follows:

(1) The lithotomy posture.

(2) The semi-inverted (Trendelenburg's) posture.

(3) The knee-elbow and knee-chest positions.

(1) Although patients placed in the lithotomy posture do not, as a rule, display any unfavourable symptoms during operations under general anæsthetics, a few remarks on exceptional cases may perhaps be made. In very obese subjects, and in those with wholly abdominal respiration, the flexion of the thighs upon the abdomen may have the effect of driving the intestines towards the diaphragm and preventing the free descent of this structure. Care must therefore be exercised in adapting any appliance such as a Clover's crutch in these cases; and should its application caused any marked respiratory embarrassment, it will perhaps be advisable for assistants to hold the patient's flexed knees apart in such a way that the thighs do not touch the abdominal walls. When Clover's crutch is used, it should invariably be adapted so that the neck-strap passes obliquely across the back of the neck, one end of the strap being made to pass under one shoulder. There is often a tendency, when the patient is placed in the lithotomy posture, either for the head to become acutely flexed upon the sternum or for it to be extended; but by a little management such a tendency, the disadvantages of which have already been fully considered, will be averted. The neck-strap and pillows should be so adjusted that, as far as is possible, the head retains its natural position with regard to the trunk. Very serious and sudden syncope has been known to occur in excision of the rectum or lateral lithotomy, from the sudden loss of large quantities of blood; and we are informed by Mr. Cripps that, recognising the importance of keeping the head low in excision of

the rectum, it is his practice to have the buttocks well raised by an appropriate pad, so that the trunk and head of the patient are on a somewhat lower level than the pelvis. This particular posture, which can be adopted without flexing or extending the head to any inconvenient degree, has the additional advantage of lessening the tendency to hæmorrhage from the valveless abdominal veins. A real though very remote danger of the lithotomy posture, is the possibility of injuring brittle and atheromatous arteries, in old and feeble persons, by prolonged and acute flexure at the knees. We are informed by Mr. Treves that a case has been reported to him in which gangrene of the leg followed the use of a Clover's crutch in such a subject.

(2) By the semi-inverted posture, in the strict sense, is meant that in which the whole body, retaining the usual relations of its parts, is sloped head downwards, so as to make with the horizontal plane an angle of about 45°. Such a posture is obviously a very difficult one to put into practice, except in the case of infants and young children, whose bodies can easily be made to lie in this position by arranging a graduated system of pillows from the neck to the feet (see Fig. 13). In the case of adults, Trendelenburg's posture, which is, for all practical purposes, identi-

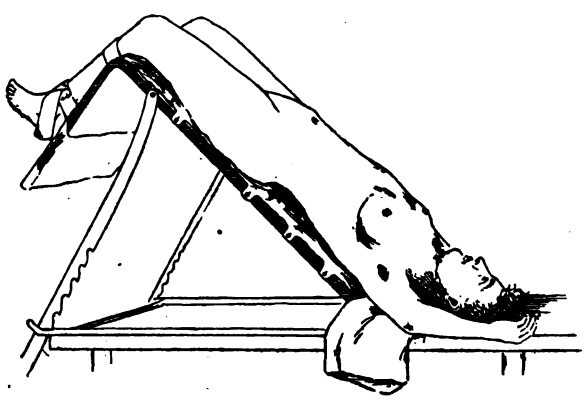


FIG. 12.—Trendelenburg's posture.

cal with the above, is the best. In this (Fig. 12) the body is placed as above described, except that the knees are flexed. This flexure of the knees to about a right angle tends to prevent the slipping of the body which would otherwise occur. Special operating tables for the maintenance of Trendelenburg's posture are now made. The patient is first anæsthetised in the customary position, and the table is then adjusted so that the body assumes the desired posture. Many surgeons regard the position of Trendelenburg as very convenient for all cases in which it is desirable to allow the intestines to gravitate towards the diaphragm away from the pelvis, and thus facilitate the removal of a deeply seated pelvic tumour, a stone from the bladder, or the return of a voluminous hernia. In such cases it may be needful to very considerably lower the upper part of the trunk. In the absence of a specially constructed table, the best plan is to raise the end of an ordinary operating table by two blocks of wood of appropriate height and with holes cut in their superior surfaces. When anæsthesia has been produced the table is tilted, and the legs of the patient are then bent over the end of the table. An ordinary chair, fixed upon the operating table so that the front edge of its seat and the top front edge of its back rest upon the table whilst its legs are turned upwards so as to form an angle of 45°, will also make a good extemporised substitute for a Trendelenburg's table. The inverted chair is covered with blankets, and the already anæsthetised patient is so placed upon it that his back is in contact with the back of the chair and his legs are flexed over the under surface of the seat. The chief, if not the only objection to Trendelenburg's posture is that in patients whose respiration is already impeded, and especially in patients with wholly diaphragmatic respiration, the pressure of the abdominal contents upon the diaphragm may lead to undesirable embarrassment of breathing. It must be admitted, however, that this does not occur to that extent which one might anticipate. We have on several occasions given anæsthetics (both ether

and chloroform) to middle-aged and rather obese subjects without this posture in any way interfering with breathing. Moreover, mucus and saliva tend to flow away from the larynx and pharynx, so that coughing, swallowing, and subsequent vomiting are to some extent averted. It should be borne in mind, at all events when employing ether in this posture, that the augmented arterial pressure may favour the occurrence of cerebral hæmorrhage in patients already predisposed to this accident.

A semi-inverted position of the head and trunk is well adapted for many operations within or about the upper air-passages, and it is not unlikely that this posture will gain ground in this branch of surgery. Thus it was adopted by Marsh[1] in the removal of a foreign body from the larynx by splitting the thyroid cartilage. The position allowed the blood to gravitate into the naso-pharynx, and thus obviated the necessity for low tracheotomy. We question whether this position has received sufficient recognition for operations upon the larynx. It is certainly a convenient one in operations for harelip in

FIG. 13.—Semi-inverted posture.

young children (Fig. 13). The child may be placed as shown in Fig. 13, or the legs may be tied together and fastened to the raised end of the table.

(3) The knee-elbow posture, which is used by obstetricians, is not often employed in this country. When it is desired to place an anæsthetised patient in this posture, which is a very inconvenient and unsatisfactory one in which to administer an anæsthetic, the patient should be

[1] 'Journal of Laryngology,' vol. viii, 1894.

first anæsthetised in the ordinary position, and then made to lie face downwards, with soft pillows under the abdomen. By a little management the weight of the trunk may be prevented from impeding thoracic expansion by keeping the elbows resting upon the operating table. We have known this posture adopted under anæsthetics for replacing an impacted fibroid, and pushing forwards and upwards a retroverted gravid uterus; but it is now rarely used.

In the knee-chest posture, which is largely employed by certain American gynæcologists, the patient kneels near the edge of the table, the arms are thrown back, and the head is turned on the side, the chest sinking down just in front of the knees. This posture is obviously only admissible when anæsthesia is not required.

IV. The Posture of the Patient after Operation.

In the consideration of the posture of the patient after operation we may say that our remarks particularly apply to the time between the termination of the operation and the re-establishment of sensibility, and that we do not propose to discuss any special postures that the surgeon may afterwards advise for facilitating dressings or drainage. We may point out that the important matter of the after-posture of cases of operation is usually entirely ignored, patients being placed in bed upon the back, with no regard to the points we propose now to enunciate and illustrate.

The application of bandages and dressings so often involves or leads to the adoption of certain postures that a few remarks may not be out of place upon this subject. Thus after breast operations it is customary to raise patients almost or completely into the sitting posture—a proceeding which we cannot regard as altogether free from risk, especially in certain cases. Should ether have been employed, and should the circulation be strong and

the colour good, there is no objection to the trunk being raised. But should chloroform have been used, and should the circulation be feeble, the body should either be raised very slightly or not at all, and any tendency to pulse failure should at once be met by lowering the trunk. Raising the body into the vertical or sitting posture is undoubtedly attended by considerable risk when there are signs of faintness. Thus a case is recorded in which a dentist attempted to administer nitrous oxide to a nervous female patient, but without success. The operation of removing seven or eight loose teeth was accomplished without any anæsthetic, and the patient fainted. She was kept upright in the chair, and the syncope proved fatal.[1] After severe and protracted operations upon the breast it is not an uncommon event for the circulation to be considerably depressed, and under these circumstances the dressings and bandages should be applied without raising the trunk. A many-tailed bandage, previously placed between the shoulders, obviates all raising of the body, and is far more convenient to adapt than an ordinary bandage.

It is always advisable to allow patients to emerge from deep anæsthesia before the thorax is constricted by bandages,—a point which not only applies to cases of removal of the breast, but to all other cases in which the thorax requires bandaging after operation.

A word may here be said as to the after-posture of breast cases. It is customary to place these patients in the dorsal posture after the bandaging is finished; but we are inclined to prefer the lateral or the dorso-lateral position, principally because mucus, saliva, and any vomited matters can far more readily escape in these postures than when the patient is horizontal, and respiration is therefore less likely to be interfered with during the recovery from the effects of the anæsthetic. We have found that patients are more likely to pass into a quiet

[1] 'Brit. Journ. Dental Science,' 1872, p. 270; 'Lancet,' vol. i, 1872, p. 727.

and refreshing sleep when placed as we have suggested than when left lying upon the back. Moreover, by arranging the patient so that the side of operation is uppermost, painful pressure is prevented.

The customary plan of applying dressings and bandages after operations for empyema also appears to us to need some modification. It is usual for the whole thorax to be bandaged more or less tightly, and for the patient to be placed in bed in the dorsal posture. Now by bandaging the whole thorax the healthier lung is prevented from expanding freely. It appears to us that some form of dressing should be applied which can be retained in position by strapping or other means. Moreover, if the patient be turned on his affected side, instead of into the dorsal position, drainage freely takes place, the dressings are kept in position, and the healthier side, not constricted by bandages, can expand with the utmost possible freedom.

There is one remaining group of cases in which the question of bandaging needs a few remarks. We refer to cases of operations upon the neck and root of the neck. It is often necessary after these operations to tightly bandage the parts, and attention should be paid to one or two points. Firstly, the patient should be allowed to come out of the anæsthesia to a considerable extent—in other words, to pass through the period of possible vomiting and coughing—before the bandages are applied. In the next place, should any impaired breathing from enlarged tonsils, adenoid growths, &c., exist, a piece of cork attached to a piece of string should be placed between the teeth to keep up free breathing. And, lastly, the patient should be turned upon the side with the head in that position which is most favourable to breathing, and careful watch must be kept over him till consciousness has returned. The tight bandaging, more especially if associated with previously impaired respiration, greatly retards the elimination of the anæsthetic; and, if the posture of the head and body be not attended

to, asphyxial symptoms may with the greatest readiness be initiated.

The position of the head with regard to the trunk is of considerable importance during the recovery from anæsthesia. The practice is too prevalent in our hospitals of sending patients out of the theatre in a semi-comatose state, with the head and trunk in those very postures which are most favourable to the development of arrested breathing. It is, therefore, not surprising that alarming symptoms sometimes arise on these occasions, symptoms which are often erroneously attributed to other causes. When the head is in the mid-line, the patient is recovering muscular power, and the ejection of mucus and saliva is about to take place, there is every chance of impeded breathing arising, more especially in certain types of subjects. Suspended respiration thus taking place is very liable to be quickly followed by pallor and other evidences of circulatory failure; and the grave mistake has over and over again been committed of supposing that the patient is in a state of faintness from loss of blood, surgical shock, an overdose of the anæsthetic or other causes. In private practice the anæsthetist and surgeon should pay special attention to these points, and should see that the patient's head is kept in that position which is most favourable to recovery from anæsthesia and to the escape of all fluids from the mouth.

Speaking in general terms, we may say that the lateral posture is preferable to all others for patients recovering from anæsthetics. Directly the administration is at an end, and the dressings are applied, the patient's body should be turned into the lateral position, his knees bent up, and his head allowed to regain its natural position with regard to the body. Without here entering into the whole question of vomiting after anæsthetics, we are of opinion that one important exciting cause of this troublesome condition is the entrance of mucus and blood into a stomach already prone to evacuate its contents. Accordingly we may enunciate a general rule, which may

thus be cited: After the conclusion of an operation, unless special circumstances forbid, turn the patient upon his side, clear the mouth and fauces of anything they may contain, and allow him to maintain this position until sensibility has returned. The side after-position is very useful for all operations attended by much after-oozing of serum or blood into the mouth, as after removal of the tongue, cleft palate, hæmorrhagic palatal or naso-pharyngeal growths, or large and vascular tonsils; and in such cases it should, in our judgment, be invariably adopted.

Children, after chloroform administration, often pass into a deep sleep which may last for an hour or more. Dr. Leonard Guthrie, in his paper on some fatal after-effects of chloroform in children, refers to the vital importance of keeping the head on the side, lest vomit or mucus enter the air-passages. "Once," notes Dr. Guthrie, "after an operation, when a child had been put to bed without these precautions being taken, I chanced to look round whilst washing my hands, and saw that she had stopped breathing. Her jaw had dropped, and she was rapidly becoming black in the face. The head was at once turned on the side, the chin was raised, and the danger was over; but in another minute it might have been too late to save her life."[1]

There are some operations after which the dorsal posture is considered essential. This is notably the case in abdominal surgery. But even in such cases it is generally possible, by placing a pillow under one shoulder and by turning the head to the opposite side, to provide for the escape of mucus, &c., from the mouth without disturbing the horizontal posture of the pelvis. This is an extremely important matter, the simple arrangement of the patient in the posture described making, in many cases, a considerable difference as regards the quietude of the abdominal contents.

After a great operation, such as amputation for railway

[1] 'Lancet,' January 27th, 1894, p. 193.

injury, the removal of an adherent and large abdominal tumour, and the like, the head should be kept low and the feet somewhat raised, the trunk being in the slanting posture. The head and shoulders should, however, be turned towards one side to facilitate as far as possible the escape of mucus and vomited material. The greater the loss of blood the greater the reason for keeping the head low—a point too often neglected in the hurry of applying warmth, galvanism, stimulants, and other active remedial measures.

It does not often happen, in modern surgery, that persistent bleeding occurs after operation; yet this troublesome condition may be met with, especially after operations upon the throat and nose,—as, for example, after removing a naso-pharyngeal growth. In such cases the posture of the patient for the first hour or so may make all the difference, for if he be kept in the sitting posture, obstinate oozing, which is often merely congestive, will very generally cease. This posture cannot, of course, be adopted should the patient display very marked symptoms of faintness. The sitting or semi-recumbent posture after such operations is more particularly appropriate when the hæmorrhage persists, and the general circulation of the patient, though possibly somewhat feeble, is not so feeble that there is risk in slightly elevating the trunk. The surgeon and anæsthetist must steer very carefully between the opposite dangers of inducing fatal syncope and encouraging bleeding from congestion in lowering the head. Raising the trunk and head may prove of great value in treating the severe venous oozing which sometimes follows operations upon vascular growths of the bones of the skull when sinuses or large diploic veins have been wounded.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. viii, p. 6.)

RENAL COLIC IN INFANTS

BY

R. A. GIBBONS, M.D., M.R.C.P.,

PHYSICIAN TO THE GROSVENOR HOSPITAL FOR WOMEN AND CHILDREN

Received August 10th, 1895—Read January 14th, 1896.

THE subject of this paper is one of which I can find no notice in the 'Transactions' of this or of any other Society. I therefore hope that what I have written may be deemed worthy of the attention of the Fellows. I have only referred to cases occurring in infants whom I have seen in private practice, and I think it better to relate these cases in the first place, and reserve any remarks until afterwards.

I have selected the following as examples of the subject of this communication.

CASE 1.—A boy æt. 10 months. Was first seen on December 3rd, 1885, because he was suddenly seized with a screaming fit, and seemed to be in great pain. The least movement evidently gave additional pain, and caused him to scream. He was lying on the arm of the nurse, on his left side, the legs being drawn up, and the thighs flexed on the abdomen. His face was flushed and expressive of terror, especially if anyone came very near him. When allowed to remain quiet in the nurse's arms his pain seemed bearable, for he did not scream. He had vomited several times.

I heard from his mother and nurse that he had been perfectly well in the morning, and that he was suddenly seized with this attack whilst being amused.

There is nothing of importance to relate regarding the previous history. He has always been apparently healthy. I could not gather that he had suffered from any previous attacks of a similar nature to the one he was then in. He was born at full time, and his mother nursed him until he was between eight and nine months old. His diet when I saw him consisted of boiled cow's milk, water, sugar of milk, and lime water, one ounce to a pint of food, and a heaped teaspoonful of sugar of milk to each bottle. In addition to this he was having two meals a day of Savory and Moore's food. This diet had been ordered some time previously, and had not been altered.

His father is very gouty, and there is a history of syphilis on his side. The mother is gouty, and comes of a gouty stock. He has two sisters and two brothers. The elder brother suffers from asthma of a severe type. The elder sister, aged sixteen, is suffering from what is probably a renal calculus, and I have attended her on three occasions for attacks of great lumbar pain, accompanied by hæmaturia, the latter usually lasting several days. The urine sometimes contains pus cells.

There is no history of phthisis on the side of the mother or father.

The child has been perfectly well since birth, and has caused no anxiety whatever.

I found on examination that the heart and lungs appeared normal. The pulse was frequent and the respiration hurried. The temperature was 102°. On having him gently undressed I noticed that he assumed the position already mentioned. He would allow me, after being present a short time, to touch the abdomen over the left flank, but if the least pressure were made on the right side in front or behind, violent pain was produced. This seemed to be especially well marked

if the left hand were placed gently over the lumbar region behind, and slight pressure made with the right hand in front, although this manipulation could be tolerated on the opposite side. It was clear from this examination that the region of the right kidney was the seat of pain. The testicle of the right side was drawn up. On looking at the urine it was observed to be perfectly clear, and the nurse stated that it was scanty in amount, and was passed in small quantities and with considerable pain. At the bottom of the vessel a few minute grains, like Cayenne pepper, were visible. A mixture containing some compound tincture of camphor, bromide of ammonium, and carbonate of lithia was prescribed, as well as a warm bath and a linseed meal poultice to the loins. The urine sent to me for examination had a sp. gr. of 1020, contained a small quantity of albumen, no sugar or bile; and, microscopically, free uric acid was found, as well as mucus corpuscles and blood. The crystals of uric acid—lozenge-shaped variety—were not very numerous.

The following morning I learned that although the treatment seemed to have caused amelioration in the urgent symptoms, the child had suffered from many severe paroxysms of pain during the night, and was still in great pain if touched over the region of the right kidney. The medicine was continued, and as the day passed the child seemed to be easier, the intervals between the paroxysms of pain becoming longer. It was now observed that the urine contained a very large quantity of uric acid, and there were many aggregations of crystals held together by mucus. This was easily discernible to the naked eye. On more than one occasion a small piece, less than a quarter of the size of a split pea, of thickish mortar-like material was passed, which proved on examination to consist of uric acid. The next morning, when again I saw the child, I heard that he had passed a disturbed night, but with few paroxysms of pain, and he now appeared much better, although he disliked to be touched on the affected side. The temperature was

normal. During the day he had hardly any pain, and the water was comparatively free from uric acid. In the evening he seemed well again.

Four months afterwards he had an attack similar in most respects to the foregoing, and three months subsequently, when seventeen months old, another one, whilst between three and four months later he had the last attack. I saw him more than three years afterwards, when he looked perfectly well, and I learned from his mother that he had been troubled with no return.

Case 2.—A boy æt. 9 months. Was first seen in December, 1886. I found him in exactly the same condition as that just related, except that the left side was affected instead of the right. He was in acute pain on my arrival, lying on the nurse's arm, on his right side, and having legs and thighs drawn up, and the left testicle retracted. He had vomited frequently. The temperature was 102·4°, the pulse and respiration hurried. He could not bear to be touched, and screamed if moved. There was nothing amiss with the lungs or heart, but it was easily discernible that the least touch anywhere about the region of the left kidney caused increase of pain. The nurse informed me that he constantly passed a little water, apparently with much difficulty and with aggravation of pain. I noticed that there were some crystals of uric acid in it, but the water was perfectly clear. On examination microscopically I observed abundance of uric acid—ordinary lozenge-shaped crystals and masses of rosettes, as well as mucus corpuscles and blood-corpuscles.

The treatment adopted was similar to that of the previous case. The time of my first seeing the child was late in the evening, soon after he had been suddenly seized. During the night he suffered from almost constant pain, but the paroxysms of acute pain became gradually less frequent and less severe; the amount of urine became greater, and a larger amount of uric acid was observable, but in the form of solitary crystals, and of masses aggre-

gated together in the midst of mucus suspended in the urine. On allowing the urine to stand in a conical glass it was noted that the sp. gr. was 1022, that there was a copious amount of uric acid, and that there were two or three minute but distinct masses, larger than the size of a hemp-seed, which could be broken down with the finger into powder. Blood-corpuscles were also observed.

In twenty-four hours from the onset of the attack the child was much better, and twelve hours later appeared well. He never had another similar attack, although I know that on more than one occasion he passed abundance of free uric acid.

This child had been fed for the first few weeks of life on Swiss milk and cow's milk, but was then changed to a wet-nurse, a strong healthy woman, who was still nursing him at the time of his attack.

In the family history I have to mention that gout is strongly marked both on the father's and on the mother's side. A brother of the child suffered from "sandy water" on many occasions. Three sisters are healthy. There is no history of phthisis on either side, and there is nothing else of importance to note.

CASE 3.—A boy æt. 22 months. Was first seen in May, 1886. At the time of my visit he was evidently in acute pain, cried continuously, and shrieked if touched. The legs were drawn up, and the thighs flexed on the abdomen. Neither testicle could be felt. It was impossible to make a proper examination, on account of the increased pain brought on by the slightest touch.

I learned from the nurse that no water had been passed for some hours, but she believed he was anxious to pass some and could not. She could tell me nothing of importance concerning the previous state of the water. The temperature was 101½°, and the pulse rapid. The skin was bathed in a profuse perspiration. I was told by the mother and the nurse that the child was perfectly well until that day, when he was suddenly seized with this acute pain.

I ordered a warm bath, poultices to the loins, and a mixture containing bromide of ammonium and compound tincture of camphor. A few hours later I saw him, when he appeared somewhat easier, but was still in much pain. I now found that this pain seemed to emanate from the right lumbar region. As in the other cases, the least pressure about the right renal region caused an exacerbation of the symptoms. On auscultation and percussion of the chest, nothing amiss could be detected. As the nurse told me that he had evidently great desire to micturate, and the bladder seemed distended, a small soft French elastic catheter was passed along the urethra without meeting with any obstruction, and a considerable amount of urine drawn off. He seemed somewhat relieved by this, and had no further difficulty in the actual passing of water, although the desire was frequent, and micturition seemed painful.

During the night he had repeated paroxysms of pain, but the intervals between them grew longer. On examination in the morning I found the same tenderness in the right flank, and the recurrence of acute pain on attempting to make pressure. The treatment was continued, and towards the latter part of the day he seemed easier. The temperature, which was 100° in the morning, became 99° in the evening. The testicles were now visible.

The following night he had some sleep which seemed refreshing. There was scarcely any pain, and in the morning the temperature was normal. He could not, however, bear any pressure over the renal region, but unless the affected region was touched the pain seemed to have disappeared.

On examination of the urine drawn off by the catheter, it was found to be clear, with a sp. gr. of 1020, and to contain free uric acid. On standing in a conical glass a copious deposit of uric acid crystals, with a considerable amount of mucus, was thrown down. There were masses of the crystals aggregated together, and suspended by mucus. With heating and nitric acid a good trace of

albumen was found; and under the microscope, blood corpuscles, mucus corpuscles, and abundance of crystals of uric acid—lozenge-shaped crystals, cubes, and large rosettes—as well as renal epithelium and crystals of oxalate of lime.

On examining the urine the following day, blood was still observed microscopically, with masses of uric acid crystals, but no fragment of anything resembling a calculus could be discovered. It appeared to me at first that something might possibly be blocking the urethra before the catheter was passed, seeing that urination was afterwards perfectly free, but nothing was discovered, although directions were given to keep all the water, and spasm of the urethra due to reflex action might, of course, account for the condition of not passing water.

The pain in the lumbar region gradually disappeared, but the urine for three days later contained masses of uric acid suspended in mucus. The albumen disappeared, and likewise the blood.

The family history is important. Gout is strongly marked, both on the father's side and on the mother's. There is one other child, an infant, who is healthy. The little patient was brought up by hand, being fed on cow's milk, and was rather rickety.

Beyond an accident at ten months old, which made him feverish for a few days, owing to a lacerated wound on the hand, there is nothing to report about his previous history. He has always been quite well. He has never had to my knowledge another similar attack, but he was much thinner after the one now described, and was some time in regaining strength, looking anæmic for several weeks. His diet at the time of the attack consisted of milk, biscuits, beef tea, chicken broth, mutton tea, and nursery pudding.

Case 4.—A boy æt. 23 months. First seen September 7th, 1886, on account of a severe attack of pain and screaming. The child was put to bed as usual, and suddenly awoke

about 10 p.m. with screaming. Nothing could pacify him, and when I arrived he was still screaming, but seemed pale and exhausted. The nurse kept him lying on her lap, but he was restless and could not be kept still. His face and body were covered with perspiration, and the surface of the body was cool. He vomited several times. The pulse was feeble and rapid, but the temperature was below normal, 97°. Beyond the great restlessness there was no difficulty in examining the chest, for the paroxysms of pain appeared to grow less, and the screaming ceased as the child became exhausted. There was nothing wrong with the lungs, and the heart sounds were merely feeble from his general exhaustion. He seemed to prefer lying on his left side with the knees drawn up. I could not palpate the abdomen, for the least touch anywhere seemed to cause great pain, and I believed that the right lumbar region was clearly the most sensitive of all. Neither testicle could be felt. He had been perfectly well until suddenly seized with this pain whilst sleeping. A mixture containing bromide of ammonium, aromatic spirit of ammonia, and compound tincture of camphor was ordered, with hot fomentations to the loins.

The next morning I heard that he had a second attack of pain, lasting a few minutes, about three hours after the first attack, and a third one about 6 in the morning, also lasting a few minutes. When I saw him he was languid, and the pulse was feeble, but he did not seem ill. He was tender over the loins, and especially over the right renal region. The testicles could be felt. The liver and spleen were not palpable. He remained languid for three or four days. The water was clear, sp. gr. 1020 to 1024, with a trace of albumen; no sugar or bile. Several specimens contained abundance of free crystals of uric acid, lozenge-shaped and rosettes, and mucus, also renal epithelium and blood-cells. In the specimen of the night following the attack a small mortar-like mass was found, which could easily be crushed with the finger, and was composed of uric acid.

The boy's diet before the attack consisted of milk, toast and rusks, milky puddings, and a little meat, chicken, or fish in the middle of the day. Before this attack he has suffered occasionally from eczema of the face and head, but from nothing else. The father is not strong, and has suffered occasionally from abscesses. He is very gouty, and comes of a gouty stock. The mother is extremely gouty, and suffers greatly from megrim and severe eczema of the body, which has proved most obstinate to treatment. There is only an infant brother.

This boy has had several subsequent attacks precisely similar to the above, always coming on without warning, and always accompanied by the passage of large quantities of free uric acid, and twice by the passage of minute mortar-like substances.

Case 5.—A boy æt. 6 months. First seen February 10th, 1887,—a brother of the above. I was asked to see him, as I happened to be in the house at the moment of seizure. He was apparently well when he was suddenly seized with screaming, and seemed to be in acute pain. Undressing him caused an increase of suffering. There was nothing to be discovered by examination except great tenderness over the left loin. Any pressure there aggravated his suffering. He screamed until he appeared perfectly exhausted. There was no rise of temperature. I noted that the testicles were retracted. He vomited frequently. When he seemed quieter and could apparently bear a little more examination he did not mind the abdomen being touched. Pressure in the left lumbar region, with one hand in front and the other behind, brought on a sharp paroxysm of pain.

The treatment was the same as for the other cases. The urine, which was difficult to collect, was acid in reaction, clear, and contained a large quantity of free uric acid and a slight trace of albumen, which subsequently disappeared. Microscopically there were blood-cells. The child remained more or less in discomfort all

day, and had two recurrences of acute pain. There was slight eczema about the scalp at the time of this attack, and three days afterwards he developed a severe acute attack of eczema of the head and face. His diet consisted of cow's milk and water, with lime water and sugar of milk.

From time to time I saw this child, and he had other similar attacks, although none so severe. He frequently, however, passed large quantities of free uric acid without any acute pain being manifest.

A sister of these children was subsequently born; and although she never to my knowledge suffered from any attack like her brothers, she frequently passed considerable quantities of free uric acid.

Case 6.—A girl æt. 12 months. First seen November 3rd, 1887, because she was suddenly seized with violent pain, screaming, and vomiting. I ascertained that she had been perfectly well, and was quite in her usual health on the morning of seizure. When I arrived she was in a paroxysm of pain and screaming loudly, and was terrified. With great suffering she was undressed. She was well nourished, and nothing could be detected amiss except that she had evidently pain about the loins. She preferred to lie on her right side on the nurse's lap, and could not bear the least touch about the left renal region, but at first examination was impossible, her contortions were so great. Afterwards she became quite exhausted and slightly collapsed, with feeble pulse, depressed temperature, and cool skin.

Similar treatment to that for the others was adopted after the patient had been gently placed in a warm bath, which seemed to give her ease. She was very depressed all day after this attack, which lasted some little time. The urine was quite clear, acid, and had abundance of uric acid. During the following night she had another severe attack, much the same as the first, lasting a considerable time, for the paroxysms of pain returned; and the following day she

passed several minute concretions of a dark brown colour. The urine also contained a faint trace of albumen; and microscopically some blood-cells were observed, as well as lozenge-shaped crystals of uric acid, and some crystals of oxalate of lime. For the next five days the child remained feeble, and then seemed in her usual health. She never had another attack like the above, but I have often seen her since, and she has frequently passed quantities of free uric acid. She was having milk, beef tea, and chicken broth for diet.

She was an only child. The father is gouty, and comes of a very gouty family. The mother is neurotic and gouty, and has a history of gout in her family for generations back.

Two years afterwards a sister was born, who proved to be a delicate child, liable to bronchial attacks, but the point of interest is that she, too, frequently had attacks of "gravel," but never such an attack as above described. They generally caused irritability, restlessness at night and by day, with general uneasiness about the loins and constant micturition.

When we speak of renal colic we usually understand that the patient is seized with severe pain in one loin, that it comes on very suddenly and with great violence, and frequently after some kind of movement, such as jumping or riding on horseback, or jolting in an uncomfortable carriage. We know that this pain, although starting from the loin and running down in the direction of the ureter, may radiate in all directions, so that sharp pain may be felt in other parts of the abdomen and down the leg. The testicle is usually retracted towards the inguinal canal, and may be extremely sensitive, painful to the touch, and swollen. The patient may be faint, cold, collapsed, covered with a clammy perspiration—in short, in real agony. Occasionally the suffering is so severe that he may be thrown into a convulsion. During an attack the pulse gradually grows frequent, small, and easily com-

pressible, whilst the respiration is also hurried. At the same time the temperature rises; vomiting is frequently met with from the very commencement of the seizure, and may continue at intervals all through the attack. The pain abates after a time, and the patient may feel comparatively comfortable, when another paroxysm of agony returns, to be followed by temporary relief or entire cessation as the case may be. During the actual seizure, the slightest movement of the body is generally followed by increase of pain, so that the patient remains as still as possible, and may assume curious attitudes in order to cause some mitigation of pain.

Whilst the attack lasts micturition is generally frequent, and the urine is perfectly clear. On the other hand, strangury may be present with bloody urine. If the kidney on the opposite side is healthy, natural urine may be secreted as usual. It must be remarked that although the urine may appear natural to the naked eye, on microscopical examination blood may be discovered, which is frequently of the greatest value in forming a diagnosis in cases simulating gall-stone colic. Cases have been reported where the urine voided has been habitually purulent and bloody from calculous pyelitis, and where, during an impaction of a stone in the ureter, the water has appeared normal, proving that the kidney on the opposite side was healthy (Ebstein, Ziemssen's *Cyclopædia*, vol. xv, p. 717, 1877).

The foregoing clinical picture is the one with which we are familiar in dealing with adults, but the cases just related are remarkable as having occurred in young infants.

Now it becomes a matter of importance to note that although the symptoms of renal colic may be present, yet no calculus may be subsequently discovered. When one is passed the matter of diagnosis is absolutely settled, but frequently it happens that this is not so, and there may be considerable doubt about the case. I mention this because, in the cases cited, there was no perfect stone discovered, although there were numerous masses of uric acid crystals.

Moreover it must also be observed that it is occasionally some days before a stone is passed after cessation of all pain or discomfort. I have a patient, a lady, who occasionally consults me regarding the passage of renal calculi, and whom I have seen in the paroxysm of pain attending the passage of a stone. She has a small box of calculi, the collection of years, of all sizes, from that of a mass one would imagine almost incapable of passing along the ureter, down to that of a mere grain. In her case it has been occasionally days after the attack before she has passed the stone from the bladder, and, what is of great importance for this paper, she has frequently not found the stone at all. On one occasion, when I attended her, no stone was passed subsequent to the attack of renal colic, but on examination of the water I found it loaded with enormous crystals of uric acid, in addition to little masses of crystals held together by mucus, and these were present for several days. There can be no doubt that an impacted stone of certain composition may become disintegrated, and be gradually washed away by the stream of urine. In such an instance it shows how important it is to keep watch upon the state of the urine if there is the least doubt about the diagnosis. And this must be of especial importance in the case of children, for they can tell us nothing of the kind or situation of the pain. It is so common for infants to have attacks of intestinal colic, that unless there is something strikingly severe not much notice is taken of the symptoms. Even in the adult, with every advantage of examination, it may be impossible to say whether an attack of acute pain, such as is usually described as renal colic, is really due to the passage of a gall-stone or renal calculus. I have myself seen one of the ablest clinical physicians we have ever had diagnose renal colic when subsequent signs proved that the attack was due to gall-stones, and it is within my knowledge that such mistakes have not unfrequently happened. Indeed, I have heard Sir William Jenner say that in some cases, when in doubt, the only way to be certain is to wait for a day or

so. If the case be one of the passage of a gall-stone, jaundice will probably appear, whereas if the pain be due to renal colic albumen will be present, or appear subsequently, in the water. In the cases brought forward blood in the urine was noticed. If then, in the adult, diagnosis between the pain due to renal colic and that due to the passage of a gall-stone be difficult, it is much more so to say in an infant what the paroxysm of pain is due to. In the cases noted it will be observed that there was evidently an increase of the pain if the loin on the affected side were touched, whilst the only position at all tolerable was when the children were lying on the unaffected side on the arm of the nurse. In the first case the slightest touch in the renal region threw the child into a paroxysm of pain, and all the movements necessary in undressing the child for examination, although most gently done, exaggerated the sufferings. It is quite evident, therefore, that the cause of pain was in the lumbar region.

In dealing with this subject it is necessary to state that in all the cases I have seen I have had no reason to confound this pain with that associated with the passage of uric acid crystals. Few practitioners of any observation can have failed to note how frequently the presence of uric acid in the urine is associated with restlessness, screaming, and general disturbance in infants. But here it is common to find sandy material in the urine if it can be saved, or on the napkins. Frequently this is sufficient to cause irritation, redness, or swelling at the orifice of the urethra; not unfrequently, indeed, if phimosis is present, in boys this urine, highly charged with uric acid, may collect beneath the prepuce, and set up balanitis, with considerable œdema of the surrounding tissues. In girls great irritation of the vulva is quite common from the same kind of urine. In association with this disturbance it not unfrequently happens that there is tenderness in both lumbar regions, and dislike to being touched at all. But I have never been struck with the fact that one loin has been markedly more sensitive than the other, although

such cases are really common, and one has ample opportunities of seeing them. They occur also at varying intervals in the same child, and the passage of uric acid may last from a few hours to several days, when the disturbance ceases. I can speak from my own experience when I say that this disturbance, caused by the occasional presence of uric acid, is most common in the children of gouty parents, and I constantly come across it. In some the passage of free uric acid is almost always present; and at this moment I have a boy aged five under my care, who has suffered for some time now from recurrent attacks of irritation caused by abundant uric acid crystals passed with the urine. This causes more or less incontinence of urine, for he frequently wets himself, both by night and by day. Yet he has no stone which can be detected in the bladder, although he has been sounded by several well-known surgeons, nor has he a stone, as far as one can judge, in the kidneys, and he has never had any attacks of renal colic. His father, as a boy, suffered from exactly the same condition, and has been subject to attacks of gout for years, whilst the father's mother suffered in the same manner. Although this case I mention is an aggravated one, I frequently have under my notice children suffering from the same kind of affection.

There can be no doubt but that the occurrence of true renal colic in infants is exceedingly rare, and yet stone in the bladder is not uncommon. Statistics with regard to the presence of stone in the bladder prove how frequently it occurs; thus, according to Dr. Prout, out of 1256 patients admitted into the Bristol, Leeds, and Norwich hospitals for lithotomy or lithotrity, 500, or nearly 40 per cent., were under ten years of age.[1]

In a paper by Mr. Morgan, published in the 74th volume of the *Transactions*, "On 114 Consecutive Cases of Operation for Calculus in the Bladder performed in the Hospital for Sick Children," twenty-nine are entered as

[1] 'On the Nature and Treatment of Stomach and Renal Diseases,' 1848, Appendix, Table III, p. 593

being of the age of three or under, the youngest being sixteen months.

Of ninety-eight calculi in children in the museum of the Royal College of Surgeons of England, kindly tabulated for me by Mr. Morison, the age is mentioned in seventy-nine cases, and of these eighteen were three years of age and under, the youngest being twelve months. In one of these cases it is noted that there were numerous small concretions, and that they were passed by the urethra. In another in the same table a small white calculus was passsed by the urethra.

Henoch says[1] that stone is just as common in children during the first years of life as in adults. Again, we know how extremely common it is for children to have copious deposits in the urine, with the least disturbance to the general system; and even in new-born children, according to Henoch,[2] uric acid infarcts are invariably present. Professor Schlossberger, who examined 199 children dying within thirty days from birth, found uric acid in the tubuli uriniferi in 32 per cent. These observations were also confirmed by Professor Martin, of Jena ('Archiv f. physiol. Heilkunde,' vol. ix, and Schmidt's 'Jahrbücher,' December, 1850, p. 333). These are gradually washed out of the tubuli, and the condition found will, therefore, depend upon the date after birth at which the examination is made, for Henoch has noted them as long as seven or eight weeks after birth. It is obvious, therefore, that with such a tendency to the formation of uric acid the chances of a calculus forming are great, whether it be found in the kidney or bladder. Uric acid concretions are the most common. They constitute five sixths of the total number of renal calculi, and wholly or in part the great majority of those found in the bladder ('Practice of Medicine,' Bristowe, 7th edit., p. 846).

[1] 'Lectures on Children's Diseases,' translated for the New Sydenham Society from the fourth edition (1889) by John Thomson, M.B., F.R.C.P.Edin., vol. ii, p. 175.

[2] Op. cit., vol. ii, p. 174.

Dr. Dickinson, in his work 'On Renal and Urinary Affections' (vol. iii), has tabulated the composition of ninety-one calculi occurring in the pathological museums of London. Of these fifty-two were simple and thirty-nine compound, and of the latter, twenty-two were composed of two ingredients. Dr. Dickinson remarks that oxalate of lime is the most frequent constituent of compound calculi, existing in about two thirds of the whole number, and that calculi, especially if they be composed of uric acid, oxalate of lime, or cystin, generally take their first concrete form in the pelvis of the kidney.

It may be presumed that the usual manner of formation is that these crystals of uric acid irritate the delicate mucous membrane of the pelvis of the kidney, causing an increase in activity of the cell-growth, and thus a greater amount of secretion,—in fact, a catarrhal condition; this secretion, covering the crystals, causes them to adhere and form little masses, and these masses, joining together and constantly being augmented, become the nucleus of a calculus. If the accumulation formed is washed away before it becomes too large to easily pass from the pelvis along the ureter, no harm is done. If, on the other hand, it cannot be washed down the ureter, it becomes impacted, giving rise to renal colic, and remains in the pelvis of the kidney, to become the source of future trouble and danger.

In the cases brought forward my belief is that the cause of the colic was still soft and friable, and in its very commencement, for under the microscope large masses of crystals of uric acid were discovered with numerous mucus corpuscles as well as blood corpuscles. It therefore appeared to me, so large and numerous were these masses—far larger than I have ever seen in the examination of the urine of infants suffering merely from what may be termed "attacks of uric acid"—that the commencing calculus had not become too hard for being crushed and disintegrated by the combined action of muscular contraction of the ureter and the flow of urine. Of

course it is possible that some small central, hardened portion or core may have passed and have escaped observation. At any rate, in these cases mentioned I did not discern anything beyond what I have described, although instruction was given to the nurse to keep a careful watch upon the urine. The fact that infants occasionally pass renal calculi is by no means rare, although that they suffer from true renal colic must be a matter of great rarity. In reference to the passage of renal calculi several well-known authorities may be quoted. Henoch mentions a case (op. cit., vol. i, p. 189) of a child five months old, who passed round fragments of the size of a pin's head, which were recognised as uric acid concretions. The case is quoted by him as an instance of reflex irritation starting in the urinary organs, for the child cried always before passing water, and developed first convulsions, and subsequently contractures of the toes of both feet, and of the fingers and knee-joints.

Rilliet and Barthez ('Maladies des Enfants,' p. 720) say that they have never met with a calculus which was impacted in the ureter, and that in their experience calculi are most common in the right kidney, where they are often large, and are rarely met with in both; but that if in both, the calculi are largely developed. They mention (p. 721) that infants may be attacked by pain in the lumbar region, or in other parts of the urinary organs, and that it may be intense and paroxysmal—a true nephritic colic.

It is remarkable that, in spite of the great tendency in infants and children to uric acid concretions and urinary deposits, so little mention is met with of true renal colic. The only explanation is that the affection must be extremely rare.

Dr. West says[1] that the formation of lithic acid in the kidneys goes on without giving rise to any very obvious symptoms, and that "he has but rarely seen a

[1] 'Lectures on the Diseases of Infancy and Childhood,' 6th edition, p. 718.

child suffer from pain of that severe character which in the adult not infrequently accompanies the descent of a calculus from the kidney to the bladder." Dr. Eustace Smith ('A Practical Treatise on Disease in Children,' 1884, p. 815) remarks that "when the concretion passes from the kidney into the ureter and downward into the bladder there is always pain, but the child suffers far less than an adult would do under similar circumstances." In a paper "On some kinds of Albuminous and Purulent Urine in Children," read in the section of Diseases of Children at the meeting of the British Medical Association by Dr. Gee, 1883, he remarked that "stone in the kidney of a child sometimes seems to be latent, and to afford no signs beyond purulent urine. In cases of this kind I do not see how a diagnosis can be made. In other cases of stone in the kidney the diagnosis becomes possible,—when there are symptoms more or less like those of renal colic; when there are symptoms like stone in the bladder, and yet no stone can be discovered; or when the kidneys can be felt by deep pressure." Mr. R. W. Parker remarked at the same meeting that the absence of what in adults was considered one of the most characteristic signs, namely, renal pain, had often struck him. He mentioned the case of a girl who had been frequently subject to periodical attacks of hæmaturia and pyuria. She had no renal colic, and during the height of an attack only complained of a little uneasiness about the umbilicus. Thus it was difficult to say in which kidney the stone was, although doubtless a stone was the cause of the trouble. This patient was subsequently operated on, and the stone discovered.

Knowing as we do the great tendency in infants and children to the formation of urinary concretions, and the rarity of renal colic, the absence of the latter may be explained by two conditions:

(*a*) That in infants the calculi do not attain a sufficiently large size to become impacted, but as a rule are washed along the ureter without difficulty.

(*b*) That the muscular tissue of the ureter is not sufficiently developed, as a rule, to cause such acute pain if a stone of a rather larger size than usual is passed.

It may also be remarked that the tissues of an infant being all lax and supple, a stone would be less likely to meet with the same relative opposition as in an adult, on account of the softness of the fibrous coat of the ureter, for we know that the fibrous structures in infants are especially soft and yielding, as is evidenced by the tendency to stretch in the various ligaments of the joints.

In adults we observe that after repeated attacks of renal colic, in some cases the sufferers do not endure continued pain from recurring attacks, and the explanation which is offered of this is that the muscular tissue atrophies, and thus the nerve supply being no longer present, pain ceases. The ureter is a muscular channel along which the calculus is forced by peristaltic action, and is composed of an external fibrous coat, a middle coat of plain muscular tissue, and a mucous lining. The mucous membrane is thin and smooth, and presents a few longitudinal folds when the ureter is open, and the nervous supply is derived from the inferior mesenteric, spermatic, and hypogastric plexuses. It is conceivable that the nerves supplying the ureter are not sufficiently developed to cause the acute pain met with in the adult by the passage of even a small stone from the kidney, and hence, in infants, the suffering is not so great; and that if, as in the cases brought forward, there is severe pain, it is exceptional, and may be due to a greater development of the nerve supply than usual, or to an inherited hypersensitiveness of the nervous system.

The subject of this paper is one which has afforded me great interest, more especially as I have met with the cases in illustration of it almost entirely in private practice. Neither at the Children's Hospital nor at the St. George's and St. James's Dispensary did I ever see a case, whilst I have only met with one in the out-patient department of the Grosvenor Hospital for Women and

Children, where I have worked for years. This to me is almost conclusive that the affection is more rarely met with in the infants of the poor, and that the cause of this is due to the greater tendency to the inheritance of gouty conditions amongst the wealthier classes, the latter being more prone to the formation of uric acid, and to suffer from conditions classed under the name "gouty."

I have not gone into the matter of ætiology or treatment in this paper, for it would make it too long, and I have merely contented myself with describing the cases. There is, of course, great variety of opinion as to the best method of treatment in those liable to the frequent formation of uric acid. I may remark, however, that in the treatment of some boys whom I have had under my care, where every facility has been afforded for the greatest care and observation, and where the diet has been recorded every day, together with the conditions of the urine for weeks at a time, I have found that an ordinary plain mixed diet has agreed best, and furnished the most satisfactory result. In one obstinate case of a boy, where I tried dieting by cutting off nitrogenous food, and placing him on milk and fish, I considered that he was worse rather than better. He continually passed large quantities of free uric acid, which caused intense irritation, so that the bladder was frequently evacuated and gave him great inconvenience. After various dieting and drugs, I found that by giving tonics, ordering plain wholesome mixed diet with meat and vegetables, and allowing him to be in the open air as much as possible, he improved more rapidly than by any other treatment. It may be noted that if infants, on absolutely a milk diet, suffer from excessive formation of uric acid, children and adults are not likely to be cured of the same tendency by placing them on milk. In view of what has been said and written on dieting in cases of excessive formation of uric acid, this point is certainly worthy of being remembered. It may teach us also that as this affection is evidently due to defective chemistry in the body, we must en-

deavour to find out to what that defect is due, and treat it accordingly.

That the subject of this paper has not previously been brought before the Society is due, in my opinion, to the fact that such cases as I have described, although undoubtedly rare, have been taken for severe cases of intestinal colic, which is, of course, extremely common.

The points to which I desire to draw attention are—

1st. That in these cases there was suddenness of onset of all the symptoms.

2nd. That there was nothing observable about the general condition of the children, and especially the urine, before the attack, to lead those in charge to seek medical advice.

3rd. That they had all the signs and symptoms of acute renal colic.

4th. That they all occurred in infants the offspring of gouty parents.

I believe I have proved in the foregoing paper that true renal colic occurs in infants; and it is possible that if what I have written leads to more careful examination of infants suffering from acute pain, more numerous cases may be discovered, and that the symptoms of infants suffering as I have described will not be too hastily attributed to ordinary intestinal colic.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. viii, p. 93.)

ON PREVESICAL ABSCESS.

BY

ERNST MICHELS, M.D.Berlin, F.R.C.S.Eng.,

SURGEON TO THE GERMAN HOSPITAL.

Received November 21st, 1895—Read January 14th, 1896.

Suppuration in the prevesical space is of rare occurrence. French surgeons have given a good deal of attention to it, especially in former times, and a few papers on the subject have been published in Germany; in this country, although the anatomical relations of the prevesical space have been admirably described, and the possibility of phlegmonous suppuration within the space frequently discussed, the clinical aspect of this condition does not seem to have attracted much attention. In the course of my surgical work I have hitherto met with four cases of this kind, and as all these cases have presented great difficulties, especially from a diagnostic point of view, not only to me, but also to the colleagues who were associated with me in the observation of the patients, I think a short account of the disease and the diagnostic and therapeutic difficulties it offers may not be uninteresting.

The prevesical space (cavum Retzii) is an accumulation of loose areolar and fatty tissue of a very elastic and yielding character, freely allowing the bladder to distend and to contract. The space is well described in ana-

tomical text-books. It is bounded (taken in the upright position of the body) above and in front by the reflection of the peritoneum, above and behind by the bladder, below and behind by the pelvic fascia, and below and in front by the symphysis pubis and the transverse fascia. To this latter point I attach some importance. French surgeons describe the space in front of the transverse fascia between this and the rectus muscle as part of the prevesical space, but anatomically and clinically this "submuscular space" is quite different from the "cavum Retzii" proper.

Abscesses occur in the "submuscular" space too, but as a rule they have nothing to do with pelvic disease; they are mostly due to traumatic rupture of the fibres of the rectus muscle, or to suppurating hæmatoma, such as occurs occasionally in the course of some infectious diseases (typhoid fever).

Many cases of prevesical abscess are described as primary, and are thought to be due to traumatic causes; but with our modern ideas on the pathology of suppuration this seems hardly compatible. There is occasionally great difficulty in tracing the origin of the suppuration, and this may have caused the authors to consider their cases primary ones. In any case the great majority of cases of prevesical suppuration are due to some pelvic or abdominal disease. Formerly diseases of the bladder and prostate gland were not an infrequent cause for the formation of prevesical abscess, but owing to the more successful treatment of those affections these cases have disappeared from the recent literature on the subject. Some years ago, at the revival of supra-pubic lithotomy, fears were expressed that the operation might be followed by suppuration in the prevesical space; but, to judge from recorded cases, this apprehension fortunately has not been realised.

Probably the most frequent cause of prevesical abscess is tubercular disease of the pelvic bones, and I have no doubt that many cases which have been recorded as

primary in reality belong to this class. In a lesser degree diseases of other pelvic structures contribute to the formation of prevesical abscess, such as parametritis, rupture of ectopic pregnancy, &c.; and two cases have been observed, one by Guyon and one by myself, in which disease of the small intestines was the primary cause.

Whatever the origin of the disease may be, its onset is a very slow and insidious one. The patient complains of a dull pain deep down in the pelvis, and suffers from great frequency of micturition, while the urine remains quite normal: there may be some rise of temperature, but this is by no means invariably the case. Pain, and the symptoms of the part of the bladder may become more and more prominent; the general health of the patient begins to suffer, and a deep-seated tumour seems to rise from behind the symphysis: if of any considerable size it resembles in its outline a distended bladder. Rectal examination will show now that the pelvis is blocked up by a hard mass closely adherent to all the bones, and the case may resemble very much one of malignant tumour of the pelvis. After a time the abscess will break through the boundaries of the somewhat narrow prevesical space, and infiltrate the whole cellular tissue of the pelvis, and subsequently may break into the neighbouring structures. Rupture into the peritoneal cavity has been recorded, but this is rare, owing undoubtedly to the inflammatory thickening of the peritoneum. More frequent is rupture into the bladder, vagina, or rectum, or the abscess may break through the pelvic floor close to the rectum, and point in the perinæum; but in most cases the abscess breaks through the fascia transversa and the linea alba, and points in the anterior abdominal wall. Cases have also been recorded and observed by myself in which the abscess broke into several of the neighbouring spaces and cavities simultaneously or successively. Some cases have been recorded in French literature of spontaneous absorption of the abscess. It must remain doubtful whether an abscess was really present in these cases, especially since

Guyon has called attention to the fact that non-purulent exudations occur in the prevesical space; he looks upon the cavum Retzii as a kind of bursa in which a hydrops is occasionally found. After some time the long-continued suppuration affects the general health of the patient, and he presents the sallow, exhausted aspect which is so regularly associated with long-standing suppuration. The final termination depends to a great extent on the primary cause of the suppuration.

While the pathology of these cases is a comparatively simple one, and while their clinical course does not differ very much from other forms of pelvic suppuration, they claim our interest on account of the great difficulties which they offer to diagnosis, and it is this difficulty, experienced in all my cases, which is my excuse in bringing them forward. At the onset diagnosis is quite impossible; the deep-seated pain, frequency of micturition, and perhaps on rectal or vaginal examination some diffuse thickening of the pelvic structures are the only symptoms, and they will hardly allow us to form an opinion with regard to the character of the disease.

Matters are still more complicated during the second stage, when a tumour has formed in the anterior part of the pelvis; then the case is in its whole aspect so much like one of a new growth of one of the pelvic structures that a diagnostic error can hardly be avoided, especially if no fluctuation can be detected and if the temperature remain normal. In two of my cases we were doubtful for a long time whether the case was not one of sarcoma of the pubic bone; prolonged observation and the use of the aspiration needle eventually led to the right diagnosis. In a third case a surgeon of the greatest eminence had diagnosed sarcoma, and had given a fatal prognosis, and from what I have seen in similar cases I am convinced that no other diagnosis could have been made. When I saw the patient a few days later, rupture through the fascia transversa had taken place; the tumour pointed in the anterior abdominal wall, showed—not very dis-

tinctly—fluctuation, and the aspirator needle withdrew pus. In my second case a correct diagnosis was not made during the patient's lifetime; the post-mortem examination revealed the true state of things. In that case which is recorded below even the aspirator needle, which otherwise is of the greatest advantage, failed to help us.

The treatment of these cases is not a very easy one, and, on the whole, not very satisfactory. During the first stage the uncertainty and difficulty of diagnosis are in the way of any efficient surgical treatment. Of course as soon as the presence of pus is diagnosed it is imperative to dissect down to the abscess, to open it freely, and to provide sufficient drainage, making perhaps, as suggested by Tillaux, a counter-opening into the vagina. If the abscess points in the anterior abdominal wall after having broken through the transverse fascia, it assumes the form of an abscess "en bissac," part of it being situated in front of the recti muscles underneath the superficial fascia, another part lying deep down in the pelvis. The communication between the two portions may be an exceedingly small and narrow one, and may be overlooked. This happened to me in one of my cases; the patient was much relieved by the first operation, but a week later all his symptoms returned. The communication between the two portions of the abscess was nearly closed, but I succeeded in finding it and enlarging it sufficiently. In one of my other cases I had to search for a long time until I found the very small communication.

In those few cases where the disease is a primary one, evacuation of the abscess and free drainage are all that is required, but in all other cases we have to look for the primary disease, and great difficulty may be experienced in tracing it, still more in dealing with it efficiently; extensive disease of the pelvic bones may be discovered, quite unamenable to any radical treatment. We may be unable to remove the source of the suppuration, and in

such cases it may spread through the pelvis even after very free opening and complete evacuation of the abscess; meanwhile the patient remains exposed to all the consequences of prolonged deep-seated suppuration. In those cases where the source of the suppuration can be attacked successfully very slow recovery must be expected; for the filling up of this large cavity by granulation tissue is a long and tedious process. This may probably be explained by the intermittent distension and collapse of the bladder interfering with the surgical rest of the parts.

I now give a short account of the four cases which I have had under my observation.

Case 1.—A. B—, admitted into the German Hospital February, 1888, a Polish Jew, whose previous history was somewhat difficult to elicit. He tells us that for some time he has been suffering from a dull pain in the pelvis, and from frequency of micturition. On admission he is found to be very much emaciated and feverish; the urine is normal. In the pubic region a somewhat indefinite swelling is found, which seems to rise from the pelvis to about two fingers' breadth above the symphysis. The tumour is very hard, its surface smooth; it resembles a distended bladder, but remains of the same size and form when the bladder is emptied. The diagnosis of a malignant tumour of the pubic bone is discussed, but under further observation the swelling enlarges more rapidly than quite consistent with a new growth, and after a little time fluctuation is discovered. Free incision and evacuation of a large quantity of pus of a very fœtid character; the exploring finger finds the bone on the posterior aspect of the os pubis roughened; repeated operations are done to remove the diseased bone. A few small sequestra are removed, and the diseased bone is thoroughly scraped; but a more extensive operation does not seem advisable. The abscess cavity closed very slowly, and the patient left the hospital much improved in his general health, but with a sinus still discharging

very copiously. He attended as an out-patient for some time, but was lost sight of finally.

Case 2.—F. D—, æt. 35, was admitted into the German Hospital on August 11th, 1888, under the care of my colleague Dr. Port, to whom I am indebted for the permission to publish the case. The patient complained of severe diarrhœa, which had been treated without benefit for several weeks. On admission he was found very cachectic and feverish; there were distinct signs of a small cavity in the right apex. The patient had uncontrollable diarrhœa, and the diagnosis was made of tubercular ulceration of the intestines. After being in the hospital for a few days the patient began to complain of great frequency of micturition; very often he had to pass water more than twenty times in the twenty-four hours, while all the time the urine was quite healthy. A few days later he began to complain of great pain in the lower part of the abdomen, and a tumour formed behind and slightly above the symphysis pubis. The nature of this swelling remained quite obscure, and an examination under an anæsthetic also failed to lead us to a diagnosis. A few days later fluctuation seemed to be indistinctly perceptible, but the aspiration needle withdrew only a little fæculent looking and smelling fluid; it was thought that the needle had been passed into a piece of intestine adherent to the abdominal wall. Nothing could be done for the patient, who sank gradually, and died six weeks after admission.

The post-mortem examination showed that the tumour in the pelvis was a fæcal abscess in front of the bladder; there was a carcinoma of the small intestine, which had become adherent to the summit of the bladder and the anterior abdominal wall; perforation of the intestine had occurred just at the point where the carcinoma was adherent to the reflection of the peritoneum; fæcal matter had leaked through into the prevesical space, and had caused

a phlegmonous abscess there. Guyon has met with a case almost exactly similar to this one.

Case 3.—B. B—, a young married woman, always in good health before, and mother of three children. Early in July, 1894, she had a sudden attack of very severe pain in the lower part of the abdomen; no medical treatment, however, was required, and after a few days' rest she felt quite well again. Six weeks later the pain suddenly recurred and was very intense; there was no rise of temperature, but the patient felt extremely ill and weak: the urine had to be passed very frequently. A few days later a swelling was noticed just above the symphysis, which increased in size during the next few days. Two medical men saw her, and came to the conclusion that it was a case of sarcoma growing from one of the pelvic structures and unamenable to operation, and gave a very bad prognosis. I saw her four days later, and the experience gained in my former cases suggested at once the probability of its being a prevesical abscess. The tumour was hard, smooth, of the shape of a distended bladder; it extended to about 1½ inches above the symphysis. Vaginal examination proved the anterior part of the pelvis to be blocked by a hard, badly defined mass in close attachment to the front and the right side of the uterus; the swelling seemed to occupy the right parametrium altogether. Fluctuation was very indistinct, but the aspiration needle withdrew pus. The next day the abscess was opened freely in the linea alba, and a great quantity of fœtid pus evacuated. While exploring the walls of the abscess cavity a small hole was detected in its lower and posterior part; this was enlarged, and was found to lead deep down into the pelvis into another cavity filled with decomposed blood and pus: the walls of this deeper portion of the abscess were very brittle, and bled copiously. The whole cavity was plugged with iodoform gauze for twenty-four hours, when all hæmorrhage had ceased, and the patient made a slow but uninterrupted recovery. From the situation

of the abscess, in close affinity to the uterus and in the parametrium, from the fact of its being a suppurating hæmatoma, and from the very brittle condition of the abscess walls, I was led to form a strong suspicion that the abscess was due to rupture of a tubal pregnancy in a very early stage, and that possibly the rupture into the broad ligament might have taken place six weeks before the present illness, when the patient complained of the sudden pain referred to above; but I am bound to say that some points in the previous history of the case did not quite agree with this supposition, especially menstruation had been quite normal and regular ever since the last confinement, two years ago.

My fourth case is still under treatment, and I am afraid will be so for some time to come, but as it presented the same diagnostic difficulties I think I may venture to add it here.

Case 4.—T. B—, æt. 35, has been suffering all his life from various ulcerations, and many operations had been performed on him; numerous scars are found distributed all over his body, one of them being due to excision of the left hip-joint, done ten years ago. There are also râles and slight dulness over the left apex.

On admission on July 16th, 1895, he seemed very weak and exhausted, was feverish, and complained of a swelling in the lower part of the abdomen; there was great frequency and pain in micturition, and in this case the urine contained a considerable amount of pus. The pelvis and the lower part of the abdomen was blocked by a hard firm mass, which on rectal examination seemed to fill up the whole pelvis, and again the presence of a large pelvic new growth seemed very probable. But when examining the patient under an anæsthetic I thought I detected fluctuation, and the aspiration needle showed the presence of pus. A large incision in the linea alba was made immediately, and a great quantity of pus evacuated; the abscess had probably broken into the bladder, for

after the evacuation the urine became soon quite clear. The bladder was washed out regularly for some time, but the fluid injected did not pass into the wound; apparently the communication between the abscess and the bladder was a very small one, and had closed very quickly. The improvement in the patient's condition was a very marked one, but it did not last long: in the first days of August the temperature rose again; pain, difficulty to empty the bladder, and diarrhœa set in. On August 6th he was again put under chloroform, and again on examination we felt grave doubts whether there was not after all a malignant new growth in the pelvis. When, however, examining the wound in the linea alba, we found that the communication between the intra- and extra-pelvic portions of the abscess which had not been sufficiently enlarged in the first operation had closed up, and it was necessary to make a free opening there. A fair amount of pus escaped again; at the bottom of the cavity the bladder was seen and felt distinctly. Free drainage was provided, and everything seemed to go on satisfactorily, when a fortnight later an abscess was seen to point in the perinæum in front of the anus; it was opened by my colleague Dr. Luce, and a large quantity of fœtid pus escaped; in spite, therefore, of free opening and drainage the abscess had broken through the floor of the pelvis close to the rectum, and entered the ischio-rectal space. Even now the recovery of the patient was only a temporary one, for after a few days of normal temperature and comparative comfort the temperature rose again and remained high, till suddenly a considerable quantity of pus was discharged from the anus. The abscess apparently has been spreading all through the pelvis, and must have caused considerable destruction, and the case seems very much like a former one, the post-mortem record of which is given by Gruber in 'Virchow's Archiv.' I may add that from the previous history of the patient we had no doubt that the suppuration in this case was due to tubercular disease of some of the pelvic bones, but we were not

able to discover the original situation of the diseased bone. The patient is just now in a fairly good condition, but still the sinus in the anterior wall of the abdomen discharges freely.

Addendum (January 16th, 1896).—I may add that in the meantime this sinus has closed, and that at the present time the patient is in a very satisfactory condition.

The more important contributions to the literature on the subject are—

Gruber, 'Virchow's Archiv,' 1862, Bd. xxiv.

Leusser, 'Archiv f. klinische Chirurgie,' 1885, vol. xxxii (gives the French literature very completely).

Pinner, 'Deutsche Zeitschrift für Chirurgie,' vol. xxiii, 1886.

Koch, 'Münchener medic. Wochenschrift,' 1887, Nos. 44 and 45.

Guyon, 'Gazette des Hôpitaux,' 1891, No. 137.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. viii, p. 98.)

ECCHYMOSES FROM NATURAL CAUSES

BY

H. A. LEDIARD, M.D.,

SURGEON TO THE CUMBERLAND INFIRMARY

Received November 7th, 1895—Read January 28th, 1896

EXTRAVASATIONS of blood within or upon the surface of the body, due to causes arising within the body, have been recognised for a long while, and if in this paper I am treading upon well-trodden ground, my reason will be found in the plea I raise for a more extended recognition of all those causes leading to ecchymoses in various parts of the tissues and organs of the body.

The subject as treated here has but one interest, viz. the part these spontaneous hæmorrhages might play in a medico-legal drama.

Death by asphyxia is caused by four well-known means, viz. suffocation, hanging, strangulation, and drowning, and if the post-mortem appearances are not constant it is due to the great variety of ways in which these four modes of death are effected.

In suffocation, for instance, there are mechanical and dynamical causes. In drowning, five eighths of the cases, according to Ferrier, are mixed, *i. e.* asphyxia is associated with other causes.

In hanging and strangulation the interference with respiration may be sudden or gradual, or the "long drop" may cause fracture of the vertebræ and crushing of the spinal cord.

These varieties in the production of the same mode of death create an inconstant variety of appearances, and tend to bring about some confusion, especially when, as in suffocation, there may be no external circumstance, such as a rope or a mark on the neck, indicating the possible mode of death.

It can hardly be a matter for surprise if these inconstant appearances in death from asphyxia do occasionally lead to judicial error.

Of the four modes of asphyxial death, suffocation is the one in which ecchymosis is the most widely distributed; and in a paper contributed by Francis Ogston ('Brit. Med. Journ.,' Sept. 26th, 1868), "On punctiform ecchymoses in the interior of the body as a proof of death by suffocation," these appearances were found on the lungs, heart, thymus, pericardium, aorta, pulmonary artery, diaphragm, pericranium, scalp, pleura costalis, liver, spleen, and kidneys. No mention, however, is made of mucous membrane.

Dr. Ogston's inquiry was not made to prove the extent and distribution of capillary ecchymoses, but to show that these ecchymoses gave strong presumptive evidence of death by suffocation.

Now the leading factors in the production of effusions of blood within and upon the surface of the body in death from asphyxia are:

1. Embarrassment or abolition of respiration.
2. Abundance of blood in the capillary system.
3. General increase of blood-pressure, greatly increased by continuous efforts to expire.
4. Gorging of the right side of the heart.

In the first instance there is general hyperæmia of the internal organs, and secondly a tendency to extravasation.

Probably no vascular organ or tissue of the body is free from this liability to ecchymosis in asphyxial modes of death. According to Briand and Chaudé (vol. i, p. 586, 10th edit.), "the diversity of the causes of asphyxia neces-

sarily determine the essential differences in the morbid phenomena after death, differences which are particularly subordinate to the more or less rapid action of the determining causes."

"If asphyxia is slow it commences by a state of agony and oppression, soon followed by swooning; the face, the lips, the orifices of all mucous membranes take a more red tint, sometimes even purple (violacée), and this coloration appears also sometimes over diverse portions of the cutaneous system." This passage is quoted because the authors (Briand and Chaudé) refer to the mucous membranes.

The length of time during which the struggle in asphyxia may be going on varies according to the means by which it is produced, and in this respect there would be a considerable difference between the instantaneous death in judicial hanging and suffocation caused by a foreign body lodging in the œsophagus or windpipe.

As a rule, two to five minutes is the duration of the struggle in asphyxia.

The extraordinary muscles of respiration are first called into action, the venous blood stimulating the nerves of the respiratory centre and evoking violent attempts at respiration. Secondly, inspiratory muscles are less active, and the expiratory muscles contract energetically. Lastly, the respiratory centres are paralysed (Dixon Mann).

The heart continues to beat for, it may be, ten minutes longer. Sensibility is soon lost, and the efforts to breathe are continued during insensibility and final convulsions.

Dr. Michael Foster sets forth how, in experiments on animals, "it is very soon observed that the expiratory efforts are exaggerated out of all proportion to the inspiratory, and how quickly they pass into convulsion. During this period (which is found to occupy a little more than a minute) the blood-pressure in the arteries rises very greatly, and the cardiac pulsations are increased in frequency." (Hilton Fagge, vol. i, p. 10.)

The morbid appearances after death from asphyxia are engorgement to a greater or less extent of the right cavities of the heart, and of all the more important viscera such as the lungs, liver, spleen, kidneys, and brain, with comparative vacuity of the left side of the heart and arterial vessels (Ogston, Lect. Med. Jurisp.).

Over-pressure in every part of the venous system is the chief factor in the production of hæmorrhagic spots noted by all authorities in asphyxial modes of death.

At first attention seems to have been fixed upon subpleural ecchymoses, with which Tardieu's name has been associated, and although they are no longer considered proof of death by suffocation, yet they are of value as a proof of death by one or other of the modes in which asphyxia destroys life. They are found on the surface of the lung when the general blood-pressure is greatly increased by continuous efforts to expire, and they are found whenever a similar relation between the respiratory movements and the blood-pressure exists. Their distribution is wider than Ogston recorded, for in hanging the mucous membrane of the stomach is not unfrequently intensely injected, a condition shared by the mucous membrane of the intestines (Dixon Mann, p. 193), and ecchymoses are occasionally found in both. Under their general remarks upon death by asphyxia, Woodman and Tidy note extravasations of blood, some minute and stellate, others of irregular form, and many of bright colour, on the mucous and serous membranes, pleuræ, pericardium, peritoneum, membranes of the brain, lining membranes of the ventricles, the conjunctivæ and the mucous membranes of the digestive and genito-urinary tract (p. 919).

In death by hanging, Ferrier remarks that vascular turgescence of the genitals in females, with sanguinolent effusion, are frequently observed. The same author notes intense congestion of the mucous membrane of the stomach.

There is variation in the post-mortem appearances in hanging and drowning, but in suffocation the fatal event

is dependent upon asphyxia alone, and the signs are more marked.

Suffocation is easy as a means for infanticide, but difficult to carry out in adult males unless stupefied. Burke is stated to have intoxicated his victims prior to suffocating them. Foreign bodies in the air passages of individuals of advanced years may cause death with rapidity, and in such the ecchymoses in the body would be but little marked.

Of all the modes of asphyxial death, suffocation is probably the one in which there would be the most prolonged death agony, whereas in hanging the compression of the blood-vessels in the neck would produce early unconsciousness. An obstruction in the air-passages or gullet whilst causing violent efforts to breathe would in no way prevent the circulation of blood in the body, but would tend to increase pressure in the venous system, and as a consequence favour the production of ecchymosis. Casper, like other authors, gives examples of capillary hæmorrhage beneath the pulmonary pleura, upon the aorta, on the surface of the heart, upon the diaphragm and tracheal mucous membrane in deaths from suffocation. In a case (No. 270, p. 187, vol. ii) of suicidal hanging, he noted "the heart completely sprinkled over with petechial ecchymoses, particularly at its base. The internal surface of the pericardium was also covered with many similar ecchymoses."

Circumstances may intervene to prevent the usual post-mortem appearances, for instance syncope in cases of hanging and drowning, and exhaustion following efforts at swimming.

When large crowds of people are gathered together, suffocations occasionally take place in public, chiefly by asphyxia, though in some cases injuries contribute to the death. In Paris in 1837 eleven men and twelve women died standing or while being borne along in a crowd. A similar calamity occurred in the same city in 1866, when about forty persons perished, the immediate cause of

death being traced to suffocation due to pressure. At Sunderland in 1883 nearly two hundred children lost their lives, for the most part by compression of the chest. The details of this disaster were noted by Dr. Lambert in the 'British Medical Journal' for 23rd June, 1883. He thought that "a hundred children died in the first five minutes after the rush occurred. In most of the cases that had advanced beyond the initial stage of suffocation we noticed convulsive movements of the eyes and limbs and almost insensibility, but it was astonishing, even in those cases where we were assured that there was only a spark of life left in them, how soon they would recover, and after a drink of water would walk away with a little assistance."

"The dead," says Dr. Lambert, "nearly all presented the same characteristic appearance, viz. a congested, puffy face, purple or blackish turgescence of the vessels of the neck, closed eyelids, protruding and fixed eyeballs, pupils dilated to the utmost, bloody froth issuing from the nose and mouth, giving the appearance of an intense degree of suffering and anxiety, yet in twenty-four hours after death much of this passed off, and the face reposed into a slight smile, as seen in sleep."

Whether the children were crushed or suffocated, or trampled down, probably makes little difference in the post-mortem appearances, as in any case respiration was more or less rapidly stopped.

From Dr. Lambert I learn that submucous hæmorrhage was present to a very large extent in every child who was crushed to death. In female children, vaginal and rectal extravasation was quite common.

In addition to asphyxial modes of death, there are certain other conditions which in the living may be associated with ecchymosis. Thus Taylor (vol. i, p. 514, 1894, 4th ed.) remarks, "Ecchymosis may sometimes proceed from causes irrespective of the direct application of violence to the skin. Strong muscular exertions, the act of vomiting, and many other conditions, may give rise to a

rupture of the minute vessels and to an effusion of blood in parts which have been stretched or compressed."

This important paragraph would have been more useful had examples been furnished of the various causes of ecchymosis; as it is, however, Taylor is the only authority who seems to recognise that difficulty might arise from mal-interpretation of spontaneous ecchymoses.

In epilepsy are seen muscular exertions of great strength, and cutaneous as well as submucous extravasations are met with. It is, again, with reference to subpleural extravasations that the ecchymoses of epilepsy have been studied, and rare as deaths actually are after a succession of epileptic fits, yet subpleural ecchymosis has been found (Briand and Chaudé, vol. i, p. 602).

In the 'Lancet' (October 8th, 1892, p. 862) a case of extensive subcutaneous extravasation of blood during an epileptic fit was noted as having been reported by Dr. Cabadé at a Congress held at Pau.

The man was a carpenter aged forty, and so severe were the extravasations that it was fully two months before the signs of ecchymosis disappeared. More or less ecchymosis from epilepsy will not transcend the observation of any practitioner, and I have actually seen it occur. A few years ago, when in the Dean Close Ward in the Cumberland Infirmary, a patient was seized with an epileptic fit; I went to the bed and saw a patch of blood appear beneath the conjunctiva of the right eye. Many of the ecchymoses of epilepsy are purely traumatic. I am not, of course, alluding to such, but in my case of subconjunctival ecchymosis the man was on his back in bed, and in Dr. Cabadé's case the man was also in bed at the side of his wife, and had no fall or any similar cause for ecchymosis.

The next example of strong muscular exertions in which it is not unusual to meet with ecchymosis of the features is difficult and prolonged labour in a strong and muscular woman.

This cause was probably kept in view by Taylor in the passage quoted, for subconjunctival and circumorbital punctiform ecchymosis is frequently met with in prolonged labour. Tarnier and Chantreuil (tome i, p. 784) remark on the presence of subconjunctival ecchymosis in labour.

Another cause of rupture of minute vessels is the act of vomiting (Taylor), and the mucus vomited is so often tinged with blood that it is quite certain that extravasation of blood may take place into the mucous membrane of the stomach. Subconjunctival hæmorrhage is also met with during strong efforts of vomiting. The straining and compression of the stomach would tend to promote ecchymosis, whilst the downward pressure of the diaphragm, often sufficient to overcome the resistance of the sphincters, might produce ecchymosis or hæmorrhage of the mucous coat of the orifices of the vagina and rectum.

In whooping cough it is not unusual to meet with ecchymoses, and the occurrence is familiar to all general practitioners of medicine.

These ecchymoses are due to the vascular turgescence of the tissues of the face from interrupted circulation during the spasm of the respiratory muscles, with perhaps further strain from vomiting.

Hilton Fagge (vol. i, p. 1020) remarks that one or both of the conjunctivæ may become ecchymosed.

Dr. Dolan (art. "Pertussis," 'Keating's Cyclopædia of the Diseases of Children,' vol. i, p. 715) remarks, "In many cases there is bleeding from the nose, ears, mouth, and rectum."

Goodhart ('Diseases of Children,' 1885, p. 206), "In many cases there are extravasations of blood beneath the conjunctiva."

Probably all writers mention this accompaniment of the disease, which is mentioned here because it further illustrates the natural production of ecchymoses from causes existing within the body.

There are several other conditions causing superficial and deep extravasations of blood, due to morbid states of the blood, such as scurvy, purpura, and scurvy rickets.

The blood extravasations of scurvy rickets between the bone and periosteum may amount to a complete bone sheath, and could hardly be attributed to external violence. In scurvy, ecchymoses are seen in various parts of the body, but especially in the lower limbs (Barlow, 'Keating,' vol. ii, p. 265).

According to Hamilton and Godkin (vol. i, p. 244, 1895), "the appearance after death of purpuric spots is very similar to that of a bruise produced at the time of death, but their size and general connection, combined with the fact that similar spots are to be found in the mucous membrane of the throat and alimentary canal, will prevent the examiner from confounding them with ecchymoses resulting from violence."

Sir Dyce Duckworth states that hæmorrhage may occur into the joints in gout, and quotes a case of Dr. Pye-Smith's where it was met with in the knees, hips, great toes, ankles, wrists, and one elbow.

Extravasations of blood into the retina in gout have been observed by Mr. Hutchinson.

In hæmophylic subjects considerable extravasations are seen from mucous surfaces.

In pernicious anæmia, gastric and retinal hæmorrhages occur, and punctiform ecchymoses have been found on the endocardium.

Capillary ecchymosis is met with in the heart and lungs after purpura, eruptive and low fevers, and cholera.

A bruised appearance succeeds erythema nodosum.

According to Carter and Frost ('Ophthalmic Surgery,' p. 529), "ecchymosis of the eyelids never occurs spontaneously except in abnormal states of the blood, as in scurvy, but ecchymosis beneath the conjunctiva frequently occurs without obvious cause. Sometimes it is produced by straining, as in coughing, or during parturition; occasionally it occurs during the night, and has

then sometimes led to a suspicion which subsequent history has confirmed, of the patient having had an epileptic fit."

Lastly, reference is made by Briand and Chaudé to the existence of subpleural ecchymosis in individuals dying of convulsions—epileptic, eclamptic, or tetanic.

There is reason to believe that in semi-asphyxial conditions and in asphyxial modes of death, the mucous surface of the respiratory passages and of the gastro-intestinal tract have received attention from those performing examinations for medico-legal purposes. The same may be said for the serous coverings of the lungs and heart as well as that of the abdominal viscera, but there is not much evidence to show that the mucous surface of the vagina has been subjected to any systematic observation. It is not possible to believe that the mucous membrane of the vagina and vulva could either by its structure or position be exempted from a liability to punctiform ecchymoses large or small, which have been noted as occurring in almost every other part of the body.

The argument would rather be that both the position and the structure of the vaginal mucous membrane would lead to spontaneous hæmorrhage in a remarkable manner.

It may probably be asserted that if there is but little record of submucous extravasations of blood in the vulva or vagina, it is not because they have not existed, but because they have not been observed or more probably because they have not been looked for.

No doubt what is chiefly required in all post-mortem examinations for medico-legal purposes is the establishment of the mode of death, and the inquiry terminates when sufficient evidence is found for the purpose held in view. Under "post-mortem appearances in death by strangulation," Woodman and Tidy (p. 949) say: "Do not forget to examine the genital organs and their vicinity, particularly in females." This passage, I take it, is rather a hint to the observer to look out for evidence of assault, and if so, blood-

stains would, to the inexperienced, at once suggest rape.

That the mucous membrane of the vagina has been observed from time to time in asphyxial modes of death is quite clear, but it has probably never occurred to any authority that the punctiform ecchymoses, if they exist, could possibly suggest rape; Ferrier, for instance, alludes to vascular turgescence and sanguinolent effusion in both sexes in hanging. Woodman and Tidy speak of extravasations of blood in the membranes of the genito-urinary tract in asphyxia, but the most remarkable confirmation of views here put forth comes from Sunderland, where, on the bodies of the female children, vaginal and rectal extravasations were quite common (Lambert).

If there is but little special material to make use of to support opinions put forward here, it must be because deaths from asphyxia are not commonly met with (if drowning is excepted), and only a small proportion of those so dying would be females, whilst a still smaller proportion would be found in such circumstances surrounding the body as might suggest rape. It is, however, just in such an exceptional case that the effusion of blood on the vulva, due to the asphyxial mode of death, might lead to error. It must be recognised that hæmorrhage upon or into the mucous membrane of the vagina may be due to natural causes, and rather than to search for proof of rape it would be the duty of the medical man to search for further evidence of death by asphyxia, and to consider whether an asphyxial mode of death was not sufficient to account for the marks of blood on the genital organs.

The circumstances of the death of a woman from obstruction in the windpipe, associated with a supposition of rape, is of infrequent occurrence, but it would unquestionably place a suspected person in a very alarming and difficult position if such charge depended entirely upon post-mortem appearances. Hence the importance of keeping in mind the possibility of the mucous membrane of the vagina sharing in the general extravasations of blood found throughout the body in asphyxial modes of death.

Rather more than two years ago an effort made to obtain the opinions of those teaching medical jurisprudence in this country resulted in some four replies out of more than a score of letters; and it would appear, though I do not say that it was so, that the attention of lecturers had not been drawn to the mucous membrane of the vagina by any special circumstance.

Dr. Dixon Mann, of Manchester, said, "There can be no doubt as to the occurrence of submucous hæmorrhages in asphyxial modes of death, and they might in certain positions be wrongfully interpreted as being due to local violence."

This opinion tolerably well sums up all that is contended for here, but such a general statement is quite insufficient for the practical working of everyday occurrences. Moreover, no such statement is to be found, so far as I know, in any English text-book upon medical jurisprudence.

It is a matter for regret that insufficient prominence has been given in works on medical jurisprudence to the occurrence of ecchymoses from natural causes, inasmuch as a tabulation of all the natural causes which may produce such appearances would be of much service to the practical medical jurist, or to those who may be called upon to perform post-mortem examinations for medico-legal purposes. In my opinion the time has arrived for the inclusion of a special article upon this subject in all new editions, for I am not sure that the importance of ecchymoses from natural causes has been fully recognised by those engaged in teaching medical jurisprudence, even if lecturers have been all along well aware of the great variety of circumstances which may lead to appearances suggestive of external violence.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. viii, p. 103.)

THE EXCEPTIONS TO COLLES' LAW

BY

GEORGE OGILVIE, B.Sc., M.B.Edin., M.R.C.P.Lond.,

PHYSICIAN TO THE HOSPITAL FOR EPILEPSY AND PARALYSIS, REGENT'S PARK

Received November 5th, 1895—Read January 28th, 1896

The statement made by Colles in 1837, that he had never seen or heard of a case in which a healthy mother had contracted syphilis by suckling her congenitally syphilitic child, has been raised to the rank of a law which goes by his name. It enjoys up to the present day universal recognition in the teaching of this country. Our authorities agree that it holds good without exception. Berkeley Hill in 1881 said[1] that "This statement of Colles made forty-four years ago, has never been controverted. Of the few cases called exceptions, not a single one bears investigation." In 1893 Mr. Hutchinson said[2] that "since Colles' day no exceptions to his law have I think been put on record which are worthy of trust." And in the last edition of Cooper's book (1895) we read[3] that "a careful examination of these cases—brought forward as exceptions—will demonstrate that owing to want of very vital points in the recorded histories, the

[1] 'Syphilis and Local Contagious Disorders,' 2nd ed., 1881, p. 53.

[2] 'Syphilis,' 1893, p. 91.

[3] 'Syphilis,' 2nd ed., 1895, p. 352.

evidence rather tends to support Colles' law than to refute it." The same view has been expressed by Hilton Fagge,[1] Lane,[2] Van Harlingen,[3] Bumstead, and Taylor,[4] &c.

A complete collection with analyses of the cases published as exceptions since 1854 does not exist. So far as I have been able to ascertain, their number is twenty. They differ in completeness, but some amongst them seem to me as conclusive as any recorded clinical facts can be. Of these I desire to submit to you two. My reason for selecting these two cases and giving them in full, is that Ranke's case has never yet been presented in the complete and convincing form in which it exists, his later report of the case having been entirely overlooked; and that Merz's case, although published in 1889 and closely approaching the exactness of a pathological experiment, has been altogether ignored. Other cases like those of Pellizzari, Zeissl, and others, are practically on identical lines with the two quoted, and their narration now would only be repetition. Behrend in 1881[5] undertook the task which I am now attempting of disproving the universal validity of Colles' law. He failed because he relied on Ranke's case in its incomplete form, and on the case of Guibout, which does not bear examination. He also erred in his historical data, as was first shown by Diday.

Ranke's Case[6]

The father, 30 years old, infected about eleven years ago, has been without symptoms for nine years. Married three years

[1] 'The Principles and Practice of Medicine,' vol. i, 2nd ed., 1888, p. 330.

[2] 'Lectures on Syphilis,' 1881, 2nd ed., p. 59.

[3] "Venereal Diseases and Syphilis." 'The International Encyclopædia of Surgery,' ed. by John Ashurst, vol. ii, Lond., 1882, p. 558.

[4] 'The Pathology and Treatment of Venereal Diseases,' 5th ed., 1883, p. 807.

[5] 'Berlin. Klin. Wochenschrift,' pp. 126 and 127, 1881.

[6] Ranke (Gröningen) "Ueber Ansteckung der Mutter mit Lues hereditaria durch ihr eigenes Kind," 'Tageblatt der 51 Versammlung deutscher Naturforscher und Aerzte in Cassel,' 1878, No. 4, p. 94.

ago, he begot in the first year of his married life a syphilitic child, which recovered after repeated calomel treatment.

The mother remained healthy until after the second confinement. The second child, born at the end of the third year, developed in the second week a macular exanthematous rash and marked ulcers at the angles of the mouth. Whilst suckling this child a small excoriation on the left nipple developed into a typical hard chancre, followed by a roseolar eruption of great severity. Inunction treatment effected a cure for the time; a relapse was treated in the same way.

During the time of observation, from the seventh month of the second pregnancy, no symptoms of disease were observed, either in the husband or in the eldest child. No milk had been drawn off by a third person.

It is, therefore, certain that the healthy mother of a syphilitic child was infected with syphilis, and with a probability approaching certainty, by her own child, while suckling it.

The case will be given more in detail at another place.

This promise was not fulfilled until 1883, when the following additional communication was made.[1]

The diagnosis of hereditary syphilis in the first born child rests exclusively on indirect evidence (report of the father, and the production of the prescriptions given for the child by the doctor formerly in attendance). This child is said to have had, besides other obvious manifestations of syphilis, ulcers at the angles of the mouth. It was not, however, suckled by the mother, having been brought up by bottle.

Before the birth of the second child a few small, slightly sensitive glands could be felt in both axillæ of the mother, which seemed to be connected with a slight intertrigo below the pendulous mammæ. A very careful examination, made during pregnancy, gave absolutely no support to the diagnosis of syphilis. After the appearance of the hard chancre on the nipple, the glands in the left axilla enlarged into a considerable mass. By this time painful swellings of the joints appeared with rather high fever, which at first made one think of an onset of acute articular rheumatism. With regard to the successive development of symptoms, the further course, however, was throughout typical,

[1] Paul Michelson, "Zum Kapitel der hereditären Syphilis" (Einiges Nähere über den Fall Ranke's), Unna's 'Monatshefte für praktische Dermatologie,' Band ii, p. 366, 1883.

and showed that one had only to deal with the prodromal symptoms of the eruption of a papular syphilide, appearing first on the surface of the chest and belly and spreading nearly over the whole body.

In the discussion which followed Ranke's communication, the late Dr. Busch, Professor of Surgery in the University of Bonn, reported a case of the same kind.

Merz's Case[1]

M. B—, 34 years old, contracted syphilis at the age of twenty-nine The disease developed in the ordinary way, and three years after its appearance M. B— believed that he might marry without danger as he had not suffered from any specific accidents for six months. Two months after his marriage he had on the tongue and in the mouth a fresh outbreak of mucous patches. Being very attentive to the appearance of the least symptom, very careful with regard to himself, besides being very anxious to have healthy children, he at once submitted himself to the "mixed treatment," and at the same time he took all the necessary precautions to avoid the communication of the disease to those surrounding him. Since that period he has never had any relapse; he has, nevertheless, continued the treatment at regular intervals. In December, 1887, six or eight months after the attack in question, his wife became pregnant. The pregnancy progressed in the normal way, and on the 1st October Madame B— was delivered, at the full term, of an apparently healthy and well-nourished boy. I must add that at the request of M. B— I had on several occasions examined his wife—who thoroughly grasped the situation—and that I had never seen anything which could lead one to suspect infection. I also examined the child at the time of its birth, and naturally ordered suckling by the mother.

All went well during a fortnight, till the parents, who at my advice examined carefully every day their child at every aperture, discovered one morning a slight excoriation on the upper lip.

Being called the same day, I found a superficial abrasion, opalescent, of the diameter of a large bean, and much resembling a mucous patch. I warned the parents that this might perhaps be the first manifestation of specific disease, but as I counted

[1] Merz, M., "Exception à la loi de Baumès dite loi de Colles," 'Bulletin médical de l'Algérie,' 1re Année, No. 1, pp. 16—26, 1889.

upon the maternal immunity, I reassured the mother as to the risks of a possible infection, and I strongly urged her to continue suckling as if nothing existed. I little suspected that I was going to have the sad privilege of witnessing an infringement of the law. During the following days other patches appeared on the lips, the tongue, and the internal surface of the cheeks of the child, together with a circumscribed eruption of pemphigus on the legs and hands. On the appearance of the first symptoms the mother had been subjected to treatment, and everything disappeared at the end of ten days. About a month afterwards, upon meeting the father, he informed me that for a week his wife had suffered from very painful fissures in the left nipple. I prescribed frequent applications of a solution of cocaine, the pain diminished, but the fissures persisted. I visited the mother eight days afterwards, and found all around the nipple ordinary cracks, but on the left side a rounded cup-shaped erosion of little depth, about the size of a five-franc gold piece, ham-coloured and iridescent on the surface, slightly painful, bleeding rather easily, but not discharging pus. On palpating the ulcerated part between the thumb and forefinger, I thought I could detect a vague induration. In the axilla there were some small hard glands moveable under the skin. As the patient was undergoing the "mixed treatment," I had nothing else to do but to suspend my diagnosis and watch her. The further development was typical. The ulceration developed exactly like a chancre. It healed in a fortnight and was followed after thirty days by a splendid roseola, accompanied by loss of the hair and by a mucous patch on the inner surface of the right cheek. I may add now that no other accident followed, thanks to a vigorous treatment. Fortunately the milk did not dry up, and the child continued to be suckled. Both are or seemed to be actually cured.

In this case the medical adviser, convinced of the practical teaching of Colles' law, acted upon it with the unfortunate result of producing just what the law pretends to guarantee against.

Cases like these must be rare even if the fact which they establish were of more frequent occurrence than it seems to be. To give them the conclusiveness which they possess, several conditions have to be fulfilled, which for

obvious reasons cannot often all be substantiated in an individual case. These conditions are the following:

1. The previous disease of the *father* has to be established.

2. It has to be proved by *medical evidence* that the disease of the child is syphilis. All cases have to be dismissed in which the previous history of the child as regards symptoms rests upon the evidence of relations, &c., because some non-syphilitic diseases of infancy closely resemble congenital syphilis.

3. It must be established that the disease of the child is *congenital* and not acquired. In this respect we have only one but an all-important point for guidance—the time of the appearance of the disease in the child, as the primary sclerosis might be missing or overlooked, and as in every other respect the acquired disease might closely mimic the congenital form. But even suppose that the child is infected at the day of its birth, taking for the first and second incubation the shortest possible intervals of two weeks and three weeks respectively, it could not present a syphilitic skin eruption, and an affection of the mucous membrane of the mouth due to acquired syphilis before the sixth week. Therefore only those cases can be accepted as conclusive in which the general syphilitic disease of the child appeared within the first five weeks. Practically, however, the outbreak of secondary symptoms up to two months or more after birth can be attributed to the hereditary form of the disease.

4. It must also be certain that the ulcer appearing at the mother's breast is a *primary* sore, and here again we have only one criterion to rely upon, viz. that the indurated ulcer is followed in due time not only by glandular swellings in the axilla, but by a rash, throat affection,—in fact, by the ordinary sequelæ of the disease. It is well known that in people with latent syphilis irritation of the skin may produce ulcerating sores. These sores may become indurated, as is the case, for instance, in the relapsing chancre, which, although occasionally

appearing without any appreciable cause, might follow irritation or herpes of the prepuce. Therefore the fact that the ulcer on the mother's breast is indurated, does not in itself prove its primary character any more than the want of induration would disprove it. Swollen glands in the axillæ might follow any ulcerative process of the mammæ whether syphilitic or not, and if syphilitic whether primary or not. The incisive difference between the irritation ulcer and the primary sore is that the former is not followed by secondary symptoms.

5. We have further to ascertain that the mother has not suckled any other child than her own, nor been exposed to contracting the disease from any other source. About these two points, in private practice particularly, information can be obtained which is not subject to any reasonable doubt.

That these conditions should all be fulfilled in the same case must evidently be rare. If it happens, an exception to Colles' law is established.

But from another point of view also the comparative scarcity of conclusive cases is easily explained. I do not think that any one now-a-days doubts that a child affected with congenital syphilis may communicate the disease to a healthy wet-nurse by suckling, but about the frequency of this occurrence the greatest diversity of opinion exists. It appears that Colles considered accidents of this kind by no means rare. Continental syphilographers have described epidemics of the disease spread in this way. On the other hand, a man of such large experience as Henoch[1] confesses to never having met with a case. Only quite recently Mr. Parker[2] reported his own experience to a like effect from the Shadwell Hospital for Children. According to Mr. Hutchinson[3] syphilitic infants when put to wet-nurse so

[1] New Sydenham Society, vol. cxxv, 'Lectures on Children's Diseases,' vol. i, p. 119.

[2] 'Brit. Med. Journ.,' February 9th, 1895.

[3] 'Syphilis,' 1893, pp. 94, 95.

rarely communicate the disease that some surgeons to lying-in hospitals with very large opportunities for observation have expressed disbelief as to its possibility.

This discrepancy cannot be satisfactorily explained by the fact alone that in different countries, and in the same country at different times, the proportion between children brought up by hand and those brought up at the breast shows great variations. It is partly accounted for by the circumstance that the syphilographer's experience must necessarily be at variance with that obtained in lying-in hospitals, children's and foundling hospitals. A collective investigation, embracing the institutions just named, could alone settle this open question, which the experience of no single man, however vast it be, can satisfactorily answer.

If infection of a nurse by suckling is rare, and if the above conditions have to be fulfilled to establish conclusive cases of infection of a mother by her own child, their comparatively small number is fully accounted for. Their number would probably be larger if the opportunity of closely watching mother and child for several months, in hospital practice at least, would offer itself more frequently; and it would be still larger if some cases had not been excluded and been put down to acquired syphilis in the child, only for the reason that those who had the chance of observing them were imbued with the teaching of Colles.

If Colles' law were valid it would have an important practical as well as theoretical bearing. If it is only the expression of the personal experience of some at variance with that of others, the practical as well as the theoretical conclusions fall to the ground.

Practically Colles' law teaches that the healthy mother can suckle her own congenitally syphilitic child with impunity. The so-called exceptions teach that she cannot do so, and that therefore she, no more than a hired nurse, should be allowed to expose herself to the risk

which she might run. In some particular case it may be justifiable to put a congenitally syphilitic child to the breast of a healthy wet-nurse who has been informed of the danger she runs. So it may be justifiable occasionally not to prevent a healthy mother from suckling her syphilitic infant. In cases of this kind, a conflict of various interests exists which cannot be settled on purely medical lines. Moral, legal, and sentimental considerations determine the decision to be arrived at. From a medical point of view no justification exists.

The *theoretical* consequence of Colles' law would be that a mother who has given birth to a syphilitic child is immune against infection by it, although she has no symptoms of the disease and never had any. This immunity has been regarded as proving the existence of latent syphilis in the mother. With what we know now on the relation between disease and immunity such an argument is not admissible. In fact, the present phase of bacteriological knowledge seems to readily offer a theoretical explanation drawn from analogy. If the normal placenta represents a filter between the circulation of the mother and that of her child for all formed substances, the passage of microbes from one circulation to the other through the placenta (at one time regarded as impossible, but now definitely proved) presupposes damage to the cell layers. On the other hand the noxious effect of the toxines produced by the organisms and carried by the blood might pass from one circulation into the other by chemical interchange. Applying this to the question of transmission one might argue that infection only takes place through the microbes and the lesions they cause, while the immunity is established by the toxines and their diffusion through the blood. Simple and plausible as such an explanation might sound it is mere speculation, and at the same time insufficient, because, apart from other things, it leaves unexplained those cases in which neither infection nor immunisation takes place. Such cases occur not only in syphilis, but

also in other transmissible diseases. The child born healthy of a mother affected with smallpox can contract the disease during parturition or in early extra-uterine life, and must therefore be protected by vaccination.[1] Vaccination of pregnant women with vaccine lymph produces immunity in the child against vaccination in at most only one third of the cases.

A certain liking for symmetry and the rounding of theories has evidently given rise to the extraordinary pendant to Colles' law which is known abroad as Profeta's[2] law, and which says that a child born healthy of a syphilitic mother is immune against its mother's disease, and can therefore be suckled by her with impunity. Going a step further it has been contended that a healthy child is immune against infection by the secondaries of its father, if the latter was syphilitic at the time of generation. That exceptions to Profeta's "law" exist was admitted

[1] Transmission of smallpox from mother to fœtus is of an erratic character. The child may be born dead with or without smallpox. The child may be born alive with smallpox. The child may be born apparently healthy, and develop smallpox shortly after birth (say two days); in this case also the disease was contracted *in utero*. The child may be born healthy, and either remain healthy or contract smallpox in extra-uterine life; in the latter case it nearly invariably dies; to protect it it has to be vaccinated, which can be done successfully. The child may, during an epidemic, contract smallpox *in utero*, the mother, who was vaccinated previous to gestation, escaping. Of twins one only may be born with smallpox.

It seems that while intra-uterine transmission of smallpox is by no means rare, intra-uterine immunisation seldom if ever takes place. The two antagonistic statements to be met with, that if a pregnant woman suffers from smallpox her fœtus will also be affected, and that the child is always born free from the disease, are both wrong. They are merely expressions of personal experience and knowledge. Smallpox occurring during pregnancy is in a large percentage of cases instrumental in producing abortions and still-births. In the majority of such cases the fœtus shows no signs of actual or past disease. Still, eruption or scars indicating intra-uterine infection may be found on the dead fœtus, as well as on the living child, at the time of delivery. The child, if born free from the disease, and remaining healthy, is neither refractory to smallpox nor to vaccination.

[2] 'Trattato Pratico delle Mallattie Veneree,' Palermo, 1888, pp. 980, 981.

by him, and also by Diday when the latter gave the law its name. Since that time other observers have brought forward exceptions. In fact, I am not sure that they are less in number than those examples which might be adduced to support this so-called law. Anyhow, we know that a healthy child born of a syphilitic mother can contract syphilis from her in its extra-uterine life, and it naturally follows that the child has to be protected against this possible source of infection. That in face of these granted facts one could act and teach others to act upon the rule, affords a remarkable instance of conviction obscuring judgment.

It is still possible that within certain limits Colles' law and its pendant express a clinical fact, viz. that under unknown conditions during gestation a reciprocity is established between fœtus and mother with regard to conveying immunity. The frequency with which this takes place has still to be found. But whatever the proportion may turn out to be between those cases which at present are considered exceptional, and those cases which would exemplify the rule, our practical dealing is determined by the former.[1] The practical consequences of

[1] The case is similar to that of vaccination syphilis. Vaccination syphilis, although fully established, is rare. Amongst the cases published as such there are undoubtedly many in which products of hereditary syphilis, eruptions following ordinary vaccination, non-syphilitic induration changes in the vaccine pustules have been mistaken for vaccination syphilis. On the other hand there is no doubt that for obvious reasons it has been thought desirable to suppress occurrences of this kind. If these two considerations to a certain extent balance each other, the fact still remains that vaccination syphilis is rare. Only in this way can it be explained that men with unusually large opportunities for observation confess to never having seen a case of this kind. No other explanation can be given for such a fact as that reported from the Vienna Foundling Hospital, where, amongst more than 100,000 cases of vaccination, extending over thirty-five years, not a single case of vaccination syphilis occurred, although little or no care was taken to prevent such accidents. Only in the same way can be explained the long array of negative evidence which clinical as well as experimental experience has furnished with regard to the vaccination of healthy from syphilitic subjects. Nevertheless, all those who grant the existence of vaccination

the law break down since the exceptions are established. To ignore the exceptions or disprove them, and act upon the rule, is one thing. To accept the exceptions, and put aside the rule, is another thing. But how anyone can accept the exceptions, and act upon the rule, like Fournier[1] and others, I fail to see.

In accordance with the actual wording of Colles' law I have restricted myself to the consideration of cases where the source of the mother's infection was her own child. No facts exist to show that a mother immune against her child's disease can still contract the disease from other sources. Granted that immunity against the child's or the mother's disease respectively means immunity altogether, then the rules referred to can in a more concise form be thus expressed:

1. A healthy mother who has given birth to a congenitally syphilitic child is immune against syphilis.

2. A healthy child born of a mother suffering from acquired syphilis is immune against syphilis.

With regard to this wider conception of what is understood as Colles' law, its fallacy is still more completely shown by those cases in which the mother of a congenitally syphilitic child contracts fresh syphilis shortly before or after delivery. Against this positive evidence the few negative inoculation results have no weight.

All the exceptions to Colles' law in its wider sense are conclusive examples of a purely paternal infection of the fœtus presenting the following features: acquired syphilis in the father, congenital syphilis in the child, acquisition of syphilis by the mother shortly after delivery.

syphilis agree that by these exceptional occurrences our practical dealing with regard to vaccination has to be guided.

[1] Fournier's position in this question is all the more surprising, as it is in open contradiction to what he argues and advocates in vaccination syphilis. With regard to the latter he emphasises that "all the negative facts in the world could not outweigh one positive authentic fact," and from the positive facts he draws the practical conclusions. With regard to Colles' law he accepts the positive facts observed by others, but he fails to draw the unavoidable conclusions.

In conclusion, and in connection with the subject of this paper, I beg to lay before this Society the following suggestions, which might, perhaps, advantageously form the basis of a collective investigation.

1. Collect cases in which a congenitally syphilitic child has infected its own mother by suckling.

2. Collect cases in which a healthy mother who has given birth to a congenitally syphilitic child, has been infected with fresh syphilis from some other source shortly before or after delivery.

3. Collect cases in which a healthy born child has contracted syphilis from its mother, those cases being excluded where the mother's syphilis was acquired after the birth of the child or too late in gestation to be transmitted.

4. Collect cases in which a child born healthy of a syphilitic mother contracts syphilis from some other source.

By the publication of Dr. Coutt's [1] case, although it is, as I have stated elsewhere,[2] not quite conclusive, the first impulse was given in this country to critically approach the traditional teaching on this question. It has furthermore already elicited from Mr. Parker [3] the information that his own experience furnishes him with at least one exception to Colles' law. A more extended inquiry is sure to produce further results.

[1] 'Lancet,' June 9th, 1894.

[2] 'Lancet,' June 23rd and July 28th, 1894.

[3] 'Brit. Med. Journ.,' February 9th, 1895.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. viii, p. 106, also at p. 155.)

THE PARASITE OF MALARIA

AS OBSERVED IN THE MALARIAL FEVERS OF THE SOUTH OF SPAIN

BY

ROBERT J. MARSHALL, M.B., C.M.,

AND

GEORGE THIN, M.D.

Received November 5th, 1895—Read February 11th, 1896

LAVERAN'S discovery of the hæmatozoon of malarial fevers has suggested many important problems in connection with the pathology of paludism, amongst which perhaps the most urgent is the question of the existence and distribution of varieties or species of the parasites which are associated with corresponding forms of malarial infections. Of these types, two have been studied and described by Golgi, who has shown that quartan and tertian agues owe their differences to distinctive varieties of the parasite, the parasite of quartan fever requiring three days, and that of the tertian fever two days, for its development. Although Golgi's discoveries have not yet received universal recognition, they have been accepted by the great majority of those who have

seriously worked at the subject, and they have naturally led to long investigations regarding the nature of the parasites associated with those other types of malarial fever which differ in their course from the classical quartans and tertians. Marchiafava, Celli, Bignami and Bastianelli, Grassi and Feletti, and other Italian observers have recorded many remarkable observations regarding the parasites found in the severe forms of fever observed, more particularly in summer and autumn, in the South of Italy. The fact that the parasites of these fevers are not usually found in the sporulating form in blood taken from the finger has, amongst other causes, increased the difficulty of the study, and although the Italian physicians have succeeded in establishing many important facts in connection with them, there are other points regarding which our knowledge is very defective.

As malarial fevers assume a variety of types in different parts of the world, and as at least three—some authorities would say five—varieties of parasites have already been found associated with as many distinct types of fever, it appears probable that as many varieties of parasites exist as there are varieties of fever, and there is thus opened out before us a large field for observation in undertaking the study of the parasites found in the blood of malarial patients. Before this necessary labour is completed much work must be done and many workers are required.

The following paper is offered by the authors as a contribution to this aspect of the subject. They are not aware that the malarial fevers of Spain have yet received special study; and they make no pretention to anything like an exhaustive study even of these fevers. By a close observation of a small number of selected cases of the fevers of the Province of Huelva they are able to put on record a certain number of facts which they hope may render their contribution, fragmentary though it is, of some value as a basis for more extended investigation.

For the more easy understanding of their record they ask that the following facts may be borne in mind.

When the young spore or amœba of the hæmatozoon attacks a red corpuscle, it is seen in the fresh blood as a small, clear, colourless object, with or without amœboid movements. As it grows, minute pigment granules are observed in its substance, frequently in active movement, and presumably by their movements indicating currents in the protoplasm of the parasite. The pigment observed in these parasites has characteristic marks in the recognised varieties, its coarseness or fineness and mode of arrangement being fairly uniform for each variety. The pigment therefore becomes useful as a diagnostic index. As the parasite develops, the red corpuscle, in some forms, alters in size and in colour, these changes being characteristic of the variety of the parasite. The reproduction of the parasite is effected by division into segments. The number of segments, the time required for their development, their relation to the size of the corpuscle, and their presence or absence in blood taken from the finger,—their absence in the blood from the finger coinciding with their presence in the blood from the spleen,—afford other important diagnostic marks. In most of, if not all, the varieties of the parasite there is found in the blood from the finger rounded or ovoid bodies of various sizes, penetrated in all directions with pigment granules, and without any trace of spore formation. These are generally recognised as sterile bodies in which reproductive development has not taken place. Small rounded clumps are also found in all the varieties, with adherent portions of pigment. The nature of these objects is not always clear, but in many cases they are undoubtedly from the centre of the sporulating body, the débris left after detachment of the spores. Pigment-bearing leucocytes are also found in malarial fever in blood taken from the finger, and the time after the attack during which they are found has also a special relation to the variety of the parasite, affording another diagnostic mark. One of us is disposed to attach some importance for diagnostic purposes in fevers hitherto little studied to

the shape assumed by the pigment in the leukocytes, which seems characteristic and uniform for at all events some varieties.

The clinical observations recorded in this paper were made exclusively by Dr. Marshall, who practises medicine at Rio Tinto, in the province of Huelva, in the south of Spain. At certain seasons malarial fever is very prevalent over the whole of that district, and in some parts, which are otherwise healthy, constitutes the chief cause of sickness. Statistics that have been collected show that amongst the working population, although always very prevalent, it has been much more so in those years in which the soil was disturbed by the construction of houses, dams, &c., and at one time, when medical attendance was much less available than it is now, these fevers were a common cause of death. There are traditions of certain years during which they amounted almost to a plague. Of late years the cases have been less severe and less numerous.

Simultaneously with examinations of the fresh blood by Dr. Marshall, stained specimens from the same cases were studied in London by Dr. Thin, and latterly Dr. Marshall combined the study of the fresh with stained dried preparations. It is interesting to note, as showing the diagnostic value of the microscope in this disease, that at the earlier stage of their studies, important diagnostic points could be determined by the examination of dried and stained specimens in London, and it was found possible, by observing the temperature charts, to select the special cover-glasses which were required for the demonstration of a special stage of the development of the parasite. This was rendered practicable by the time at which each specimen of blood was taken being marked on the chart with a number, the cover-glasses sent bearing the corresponding numbers. After a year spent in general preliminary study of a considerable number of cases, a series were eventually selected for close continuous observation, and from these selected cases a certain

number have been further chosen for description in this paper as illustrative of the several types observed.

The division of malarial fevers into regular and irregular, which has been made by some authors, is applicable to the fevers which are observed in these districts. The regular fevers are those in which the clinical history and temperature charts show a striking regularity in the time and occurrence of the paroxysm, in the length of its intermission, and the degree of fever, and in this class we include fevers which are recognised even by the people themselves as quartan, tertian, and quotidian fevers. The individual paroxysms in each of these fevers resemble one another so closely as to be indistinguishable one from the other, the points of difference being the length of the intermission between the attacks. Each paroxysm has the well-known cold, hot, and sweating stages, and in any given case these stages may be separately well marked, or the relative severity of the stages, one to the other, may be liable to great variations. The attack is ushered in by pains in the back, neck, and by headache, followed by violent shivering, the thermometer showing, even during the cold stage, a high temperature—104° F. or more. After a cold stage, which lasts not more than an hour and a half, and usually in a much shorter time, the patient begins to feel very hot, has severe headache, and bursts out into profuse perspiration. Then the temperature begins to fall until the patient is free from fever, and is relieved of his painful sensations, although he is weakened by the attack. The entire paroxysm will last from six to twenty-four hours, or longer in a very severe case. The patient suffers most in the cold stage. This stage may be short, and only a slight sensation of cold felt. The hot stage may be long and severe, and the headache intense. The sweating may be so profuse as to saturate the bedclothes.

The paroxysms in the quartan are probably, as a rule, shorter than in the tertian or quotidian.

We have described the chief symptoms of the malarial

paroxysm as they are observed in Spain, in order to show that they agree essentially with those which characterise fevers of the same type in other parts of the world.

Under the term irregular fevers, we comprise those in which there is no well-marked regularity, either in the time of occurrence of the paroxysm, the length of intermission, or the severity of individual paroxysms. In many cases in which the three stages of the fever are well marked, a considerable irregularity is observed in the temperature chart. Two paroxysms may occur in the same day, or the periods of intermission may be irregular. In some rare cases there is a paroxysm every ten to fifteen days. In others there is no well-marked paroxysm. The cold stage may be absent or be very slight, and the hot stage may or may not be noted by the patient. Sweating may be profuse, or slight, or absent, and one paroxysm may pass into another without any intermission. In these cases the symptoms are usually headache, aching in the bones, and general depression, the patient feeling ill and incapable of doing anything. The appetite is generally poor, and there is a more or less marked cachexia. Under this class come some of the very acute summer fevers, in which there may be no normal temperature registered for a few days, and in which the patient is quite prostrate. In this case there is generally intense headache, and aching in all the body, and the anæmia and the weakness consequent on the attack are very great. In many cases there is in addition to high fever very acute vomiting and jaundice, resembling a severe bilious attack, or there may be choleraic symptoms, which are apt to be misleading regarding the nature of the disease.

Attacks of pernicious fever in which death occurs rapidly whilst the patient is in a state of coma are sometimes found here, but they are rare.

The Season of Fever.

In this district fever begins with the hot weather, and occurs with greatest severity during the very hot months, the worst cases being from the middle of July to the middle of September. The first cases of fever show themselves in May, and the malarial season practically ends in the first weeks of October, when the hot weather is over. This refers to the time when the disease may be contracted, fevers rarely beginning during the winter and early spring in people who have not lived for a summer in the districts. A few such cases have, however, been noted. Chronic cases are found throughout the year, but can generally be traced to an acute seizure during the hot months. The length of the fever season depends on the atmospheric conditions, great heat after rains being looked upon as very favourable to the production of fever. A cold summer or one in which there is much rain without great heat is less favourable to its development. A close moist heat is more favourable to the development of the fever than a dry heat. During the colder months quartan fever is the form generally seen, and also cases of low fever associated with cachexia. A chill may provoke an attack in a person who has already had fever or who has been living in the district.

We shall now describe separately the different types of malarial fevers observed in this province, with illustrative cases.

Quartan fevers are comparatively common in this part of Spain. It has been shown by Golgi that a quartan fever is caused by the existence in the blood of a parasite which requires seventy-two hours for its development, each paroxysm being coincident with a division into spores of one generation of this parasite. In a double quartan there are two generations at different stages undergoing simultaneous development. This is the form of fever that is most prevalent in winter and

spring, and though very chronic in its course, and requiring very energetic treatment for a long time after it seems cured, owing to its great liability to recur, it is the form which produces the least ill effects in the patient, the two days' intermission between each attack allowing him time to recover a certain amount of strength before the next attack. Frequently the patient continues his work, being absent only on the day of the paroxysm, or he may work up to the very moment in which he is attacked with the cold stage.

Many patients have a good appetite between the attacks, and feel fairly well, the only noticeable symptom being a certain amount of anæmia. But in severe cases, and in double quartans, the patient feels ill during the intermissions.

Quinine in large doses in most cases cuts short the attack, but it is always necessary to continue taking quinine at intervals for a considerable time on account of the great liability to relapse shown by this form of malarial fever. The paroxysms occur with great regularity, and the patient can often tell with precision the hour in which he will be attacked. The following cases are related as a sample of the quartan fevers observed in the south of Spain.

Quartan.

Case 1.—Josè Fernandez Rosa, æt. 26, a miner, was admitted to the Hospital on November 15th, 1895. He has lived in this district for fifteen years, and was first attacked with fever two months ago, the type being quartan, and the attacks occurring with great regularity at about 4 p.m. on the corresponding days. The patient is a fairly well-built man, but very anæmic, and with a dirty yellow complexion. The spleen is enlarged and tender.

November 15th.—Temperature 11 a.m., 99·2° F.; 3 p.m., 99·6°. Attack began at 3.30 p.m. with pain in the back and

legs. 4.20 p.m., temp. 105° F.; shivering violently. 6 p.m., temp. 102·4°; sweating, and more comfortable. 12 midnight, temp. 99·6°.

Microscopical appearances seen in stained preparations.—4.30 p.m. Small unpigmented parasites in red corpuscles. 8.30 p.m. Small parasite filling one quarter of red corpuscle with very little pigment. These parasites were of probably twenty-four hours' growth, and would correspond to a period of maturity on the 17th.

16th.—Patient was up and feeling well all day; the temperature, except for slight rise to 99° F. at 8 p.m., was normal.

Microscopical appearances in stained preparations.—11.40 a.m. Small pigmented parasites filling one third of red corpuscles. Very young form free. (Two forms of the parasite, a very young form from the spores of the 15th, and a large form, being advanced growth of those observed on the 15th, were distinguished.) 5.20 p.m. Very young forms adherent to the edge of the red corpuscle.

17th.—Temperature rose after 8 a.m., reaching 101·6° at 8 p.m. The patient, however, felt well all day.

Microscopical appearances.—4.30 p.m. Parasite filling one third to one half of red corpuscle. Parasite filling two thirds of red corpuscle with pigment which is not central. These belonged to the generation which would arrive at maturity on the following day. (The sporulating forms causing the slight fever on the 17th were not observed, probably owing to the small number in which they were present.)

18th.—Temperature a 8 a.m., 98·8°. One gramme quinine sulphate given in solution at 10 a.m. 12 midday, temperature 99° F. 3.30 p.m., temperature 100·4°; 5 p.m., 104·6° F.; 6.45 p.m., 105·6°; 9.30 p.m., 100·8° F.

Microscopical appearances.—11.25 a.m. Parasite filling four fifths of red corpuscle with peripheral pigment. 3.15 p.m. Sporulating forms. (These forms correspond to the paroxysm on the 18th, the sporulating forms being

observed just before the onset of the attack.) 6.45 p.m. Skeleton of parasite with central pigment adherent.

19th.—Temperature rose in the morning, reaching 100·4° at 8 p.m. One gramme quin. sulph. was given at 10 a.m.

20th and 21st.—One gramme quin. sulph. given each morning at 10 a.m. There were, for a few days afterwards, short rises of temperature to 99° F., but the appetite continued good, and the patient was discharged well on November 28th. The bowels were costive throughout, requiring an occasional saline draught to move them.

Previous to entering the hospital he had taken quinine at intervals without affecting the course of the fever. Two weeks previous to his entrance one gramme of quinine was given daily for four days in five-grain doses three times a day, but without effect. The same quantity given in the hospital in one daily dose of one gramme effectually cured the fever. It should be noted that the first dose of quinine was given five hours before an access of fever. According to Golgi's observations, the spores of the fresh generation set free on the 18th would meet in the blood a sufficient concentration of quinine to destroy their vitality at the stage when the parasite is most susceptible to the action of the drug. On the 17th, the day before the crisis, although the patient felt quite well, the temperature rose at 8 p.m. to 101·6°, a fact that would indicate that we had probably here to do with a case of double quartan of which one generation was very weak.

The microscopical observations bear this out, because on the evening of the 15th there were found a few small parasites in a very early stage which would correspond to a period of maturity on the 17th.

That more of this generation were not observed during the 16th and 17th may be accounted for, as already remarked, by the small number in which they

were present. It may be presumed that a double quartan was just beginning.

The parasite in this case had the recognised characteristic appearance of the quartan parasite, as described by Golgi. The pigment was large and coarse, the spores were large, mostly eight in number, and converged close to a central mass of pigment, and the corpuscle was scarcely enlarged.

Case 2. *Double Quartan.*—Jose Pantiga, æt. 13, a bright, fairly well-nourished, but anæmic boy, states that he has suffered from chronic fever of a quartan type at intervals for three years.

This boy came under observation on two occasions, the first being in April, 1895. On this occasion no record of temperature was taken, but the boy and his mother stated that paroxysms occurred on the 17th and 20th April, and this entirely bore out the microscopical appearances observed each morning in fresh blood.

April 17th, 11.30 a.m.—

Microscopical appearances in fresh blood.—*a.* Sporulating parasites. In some the spores were separate and congregated round the central mass of pigment. There were from eight to ten in each parasite. *b.* Large sterile forms and débris.

These appearances showed that an attack was imminent, and according to the boy's statement it began at 12 noon, half an hour after the observations were made.

18th, 11.30 a.m.—*a.* Small amœboid parasites with a few granules of pigment in slow movement, occupying about one sixth and one fifth of the red corpuscle. *b.* Débris. This would correspond to twenty-four hours' growth of the spores observed on the 17th.

19th, 9.30 a.m.—

Microscopical appearances.—*a.* Parasite filling two thirds of the red corpuscle, with coarse pigment having a sluggish movement. (A more advanced growth of the parasite observed on the 18th: would reach its full

development and sporulate on the 20th, causing a paroxysm, and this is what occurred.) 20 grains quin. sulph. was given in the morning of the 20th at 8 a.m., and the boy had a severe attack of fever not long after. The next observation was made in the morning of the 22nd.

22nd.—*Microscopical appearances.*—*a.* Large sterile parasite. *b.* Pigment-bearing leucocytes.

23rd.—*a.* Sterile parasite.

25th.—*a.* Two parasites occupying one third of red corpuscle, with peripheral pigment granules. *b.* Sterile parasite. *c.* Pigment-bearing leucocytes.

29th.—*a.* One parasite observed occupying more than one half of red corpuscle. *b.* Sterile forms and pigment-bearing leucocytes.

May 2nd.—*a.* One parasite seen in red corpuscle. *b.* Sterile forms. *c.* Pigment-bearing leucocytes.

3rd.—*a.* One sporulating parasite observed. *b.* Pigment-bearing leucocytes.

There was a slight attack of fever on the 30th April ten days after quinine, but there was no real paroxysm experienced till May 16th, twenty-six days after.

It will be observed that 20 grains quin. sulph. given in one dose before the attack on the 20th April, whilst it had the effect of practically preventing any paroxsym till the 16th May, still was not sufficient to entirely destroy the parasites. To a great extent, however, it modified the development of those that remained, only a few parasites being observed in a natural state of development, corresponding more or less to attacks which would have occurred had the parasites been present in greater number. The sterile parasites were more abundant, showing that the quinine, though not sufficient to kill, still prevented their true development into spores.

There was throughout only one generation at a time observed in the blood, corresponding very closely in their development to the history given by the patient.

The boy was not again seen till the 20th May, when he was admitted to the hospital. He stated that he had had

fever on the 16th, and again on the 18th and 19th May, each attack being associated with severe cold stage, headache, and pains in the limbs.

This, which was the type of a double quartan, corresponded to what was afterwards observed while he was an inmate of the hospital.

May 21st.—Temp. 8 a.m., 98·4° F. Between 8 a.m. and 8 p.m. he had a paroxysm, the temperature rising to 101·8° at 2 p.m.

Microscopical appearances in fresh and in stained preparations.—8.15 a.m. *a.* Sporulating parasites, eight to ten spores. *b.* Parasites filling one half to two thirds of red corpuscles with coarse peripheral pigment. *c.* Sterile forms. 2.15 p.m. *a.* Parasite filling half to two thirds. *b.* Sporulating forms. The spores were in many instances separating from each other.

There were here two generations of parasites, one fully developed, causing the fever of that day, the other, two thirds grown, causing the next day's fever.

22nd.—Paroxysm began after noon; temp. 102·8° at 4 p.m., 98° at 10 p.m.

Microscopical appearances in fresh and stained preparations.—8.20 a.m. *a.* Sporulating forms. *b.* Small parasites with a few granules of pigment filling one sixth of red corpuscle. Two were observed filling two thirds, with peripheral pigment. *c.* Sterile parasite. 10.40 a.m. Sporulating forms. 3.45 p.m. *a.* Young parasite filling one fifth to one sixth with small quantity of peripheral pigment granules. *b.* Sporulating forms with spores separate.

It will be noticed that on this day also there were found two generations of parasites, the one at the sporulating stage causing the fever on the same day, the small forms representing the twenty-four hours' growth of the spores set free on the 21st. The large parasites in the red corpuscles were probably late members of the brood which sporulated on this day.

23rd.—Temperature subnormal all day.

Microscopical appearances in fresh and stained preparations.—8.45 p.m. *a.* Young parasites with a few granules of pigment in the periphery. *b.* Parasites filling one half or more of the red corpuscles, with peripheral pigment. *c.* Sterile forms. [The younger forms here correspond to the spores set free on the 22nd, the other and older form to those set free on the 21st.]

24th.—Temperature rose again after midday, reaching 102° at 6 p.m. Ten grains quin. sulph. were given at 2 p.m.

Microscopical appearances.—8.30 a.m. *a.* Sporulating forms. *b.* Parasite with peripheral pigment filling about one half to two thirds of red corpuscle. *c.* Sterile forms.

Thus the two generations were again found.

25th.—Attack began after noon, temperature reaching 101° F. at 4 p.m. Ten grains quin. sulph. were given at 10 a.m., and again at 10 p.m.

Microscopical appearanccs.—9.30 a.m. *a.* Sporulating forms. *b.* Sterile forms. *c.* Pigment-bearing leucocytes.

Thus only one generation was found, namely, that causing the fever on the same day, no small parasites of the twenty-fourth attack being seen. Ten grains quin. sulph. were given on the 26th and at intervals afterwards, the patient also taking arsenic in small doses daily. He was discharged well on the 1st June, having had no fever after the 25th. He was seen some time afterwards and was well, continuing to take quinine at intervals.

The blood was examined occasionally afterwards and the following appearances noted:

27th.—Two days after last attack.

Microscopical appearances.—*a.* A few sterile forms. *b.* Two parasites with a very small quantity of pigment.

28th.—*a.* Sterile forms. *b.* Two large pheripherally pigmented parasites occupying nearly the whole of the red corpuscle. *c.* One small parasite in a red corpuscle with quiescent pigment, probably a dead parasite.

30th.—*a.* One parasite seen with small quantity of pigment. *b.* One sterile form.

June 1st.—*a*. Nothing observed.

11th.—Nothing observed.

Here the clinical history and the microscope showed we had to deal with a case of double quartan. It will be seen that on every occasion in which the blood was examined on the 21st, 22nd, 23rd, and 24th, two generations of parasites were observed, one being twenty-four hours more advanced than the other.

On the 25th it is interesting to note that only one generation was found, it being at the sporulating stage, and being the parasite which caused the fever on the 25th. No young forms were found of the generation which caused the fever on the 24th. It is therefore extremely probable that the quinine given on the 24th, before the fever, had killed the spores set free on that day without affecting to any great degree the generation of two days' growth which caused the fever on the 25th. It is evident, however, that the quinine, with the ten grains given on the morning of the 25th, had the effect of arresting the growth of some of the parasites which belonged to this generation, as the fever did not rise so high nor was the attack so severe as those previously noted, and the sporulating forms found were fewer in number.

Parasites were observed on the 27th, 28th, and 30th April, showing that the quinine given up to that time had not been sufficient to entirely destroy them.

The presence of large sterile forms throughout was indicative of the fact that numbers of the parasites did not undergo their natural development into sporulating bodies and spores.

The preponderance of sterile forms over the developing parasite noted in the later observations illustrates the effect of the quinine. The fact that each sporulating parasite produced from eight to ten spores, of which, in the later days, the most became sterile bodies, would sufficiently explain their number as compared with forms indicative of progressive life. It must also be noted that

the parasites observed in April were identical in appearance and development with those found in May.

The following summary of the appearances we have observed shows how completely the life history of the quartan parasite of the south of Spain agrees with that described by Golgi as characteristic of the quartan parasite of the north of Italy.

Quartan Parasite, Hæmamœba malariæ (Grassi and Feletti).

In an early stage, when twenty-four hours old, it occupies about one sixth of the corpuscle, which is normal in size and colour. The pigment granules are coarse, and mostly found in the periphery. The parasite has a slow amœboid movement, which gets less active as the development proceeds. The pigment granules have a slow movement, oscillating, or from side to side. The more mature parasite is generally quiescent, although the pigment granules may have a slightly oscillating motion.

The pigment granules remain mostly peripheral till before sporulation, when they begin to congregate in the centre, this being the indication that sporulation is about to take place. The red corpuscles do not enlarge, and though only a rim of red corpuscle may be seen, it has the normal colour.

Sporulation takes place by the division into eight to ten large spores collected round a central mass of pigment. Each spore in fresh blood generally has a brilliant dot, which is its nucleolus. The pigment in some cases is not entirely central, a few granules being scattered between the spores.

The coarse pigment and paleness of the parasite contrast strongly with the normal colour of its host, and make it easily picked out, distinguishing it in this respect from the tertian parasite. The pigment granules are coarser and fewer in number than those found in the tertian parasite, and have not so active a motion. The

amœboid action is also less active than that of the tertian at a corresponding stage of development.

The sterile forms are usually larger than the mature parasite. Their pigment may be quiescent, this being the case if the granules are collected together in masses. In others the pigment granules are scattered evenly throughout the parasite, and have an extremely active movement which has been well compared to the "swarming" of midges. If forms in which the granules are collected together are watched under the microscope, the granules may sometimes be observed to become separate and evenly distributed through the parasite, and then become extremely active.

TERTIAN FEVER is characterised by the well-marked paroxysm which occurs every alternate day, there being an intermission of one day. The length of the intermission depends on the duration of each paroxysm, and may be more or less than twenty-four hours. In a simple tertian the paroxysm will last about twelve hours or longer, and in severe cases the intermission may not reach twenty-four hours. From one paroxysm to another the interval is forty-eight hours.

As Golgi has shown, this form of fever depends on the presence in the blood of a parasite which requires forty-eight hours for its full development, each attack being coincident with the maturation of one generation of the parasite. In this part of Spain its severity varies according to the season. In the spring and early summer the attacks are milder. They increase in severity as the hot weather goes on, until in August and September they may cause very severe and sometimes pernicious attacks. In a double tertian fever there is a daily paroxysm, and two generations of parasites are found in the blood, which reach their full development on successive days. The clinical history of such a case is identical with that of quotidian fever, and it is interesting to note that most of the cases of quotidian fever observed by Dr.

Marshall at Rio Tinto, in the summer of 1895, and in which the blood was examined, showed that he had to deal with cases of double tertian, as was clearly shown by the presence of two generations of the parasites in two distinct stages of development.

This form of fever produces a more severe effect on the patient than is the case with the quartan fevers; the intermission being shorter gives less time for recovery between the attacks, and the anæmia produced is greater. Though often chronic in its course, it is less so than is the case in the quartans, and it is much less liable to recur if it is promptly and energetically treated with quinine.

The following examples of tertian fever are related:

Case 3.—Florence Martinez Romano, æt. 20, a miner, a thin, weakly man, was admitted to the hospital on March 5th, 1895. He stated that he had had attacks of fever occasionally for a year, associated with a severe cold stage, and that for the past month he had had it at intervals, the type being quotidian. The spleen was enlarged and easily felt.

March 6th.—Temperature, 12 noon, 98° F.; 4 p.m., 104·6° F.; 8 p.m., 102·6° F.; midnight, 98·4°.

Microscopical appearances in fresh and stained preparations.—2 p.m. *a*. Sporulating parasites. *b*. Parasites filling one half to one third with peripheral pigment. 4 p.m. *a*. Sporulating bodies. *b*. Parasites filling two thirds of red corpuscle, with peripheral pigment. *c*. Parasite filling one quarter to one third, without pigment.

7th.—Noon, temp., 98·4° F.; 4 p.m., 104° F.; 8 p.m., 100° F.; midnight, 98·4° F.

Microscopical appearances.—3 p.m. *a*. Sporulating bodies and spores. *b*. Young parasites, some unpigmented and some just beginning to be pigmented.

8th.—Temperature, noon, 98·6° F.; 4 p.m., 105·2° F.; 8 p.m., 99° F.; midnight, 99° F.

Microscopical appearances.—10 a.m. *a*. Young para-

sites in red corpuscles, some double. *b*. Parasites filling two thirds of red corpuscle with peripheral pigment. Quinine sulphate, gr. 20, given at noon. 2.45 p.m. *a*. Sporulating forms. *b*. Small parasite with faint peripheral pigment. *c*. *One crescent observed.*

9th.—Temperature, noon, 98·6° F.; 4 p.m., 104° F.; 8 p.m., 100° F.; midnight, 97·8° F. Quinine sulphate, gr. 20, given at noon.

Microscopical appearances in fresh blood and stained preparations.—9.45 a.m. *a*. Parasites filling two thirds of red corpuscle. *b*. One crescent. *c*. Many (sterile) free bodies with scattered pigment. 2 p.m. *a*. Red corpuscles with small spores. *b*. Sporulating bodies with separating spores. *c*. Young parasites and sterile forms. The temperature fell at night, and did not rise again, and he was discharged well on the 24th.

Microscopical appearances.—11th.—*a*. One crescent. *b*. Pigment-bearing leucocytes.

12th.—Four crescents.

22nd.—*a*. Pigment-bearing leucocytes. *b*. Leucocyte containing a pigmented body resembling crescent. Nothing was observed between the 12th and 22nd, although the blood was examined several times.

The clinical history of this case was that of a quotidian fever, but the microscopic appearances showed it to be really a case of double tertian, two generations of parasites being found in the blood in each examination.

On the 6th, 7th, and 8th, a little time before each attack, there were found the sporulating forms causing the attack, and also the parasites in an earlier stage of development, which would cause the fever of the following day.

On the 8th, 20 grs. of quinine were given four hours before the attack. This dose ought to have killed the spores produced that day, and that they did so is borne out by the appearances noted on the 9th, when there was only observed one generation, namely that causing the attack. There were also seen sterile forms, showing the

action of quinine in preventing the natural development of the parasite.

The quinine given on the 9th destroyed the spores produced that day and cured the tertian fever, as no further rise of temperature was recorded and no parasites were afterwards observed in the blood, except the crescent form which is not connected with tertian fever, and which unfortunately is very resistent to the action of quinine.

Patient was readmitted on May 7th. He stated that he had fever the day after leaving hospital and for nine consecutive days. After this he was without fever till the 5th, when he had an attack, and again on the sixth. The spleen is enlarged and the liver large and tender.

May 7th.—Temperature, noon, 100·4° F.; 4 p.m., 104·6° F.; 8 p.m., 101·6° F.; midnight 97·6° F. Attack ended with profuse sweating.

Microscopical appearances in stained preparations.—11.30 a.m. *a.* Parasites filling one third to one half of red corpuscle with or without pigment. *b.* Parasites almost filling red corpuscles with pigment beginning to group, indicative of beginning segmentation. *c.* One crescent. 1.45 p.m. *a.* One sporulating form with central pigment and twelve spores. *b.* Parasite filling one third to one half of red corpuscle. 8.30. p.m. Parasites filling one third to one half or more, with peripheral pigment.

8th.—Temperature 8 a.m., 99·6° F.; 12 noon, 105·6° F.; 4 p.m. 102° F.; 8 p.m. and midnight, 99·6° F. Attack ended as on 7th with profuse sweating. Quinine sulphate 10 grs. given in the evening.

Microscopical appearances.—8.45 a.m. *a.* Very young parasites with or without pigment. *b.* Mature parasites with pigment combining to form blocks. 12 a.m. Half-developed parasite.

From the 9th to the 14th there were daily rises of temperature to 100° F.; on the 10th and 11th it did not fall lower than 99° F. There was severe cough and slight congestion at the base of the right lung, with inflamed and tender liver. Quinine sulphate 25 grs.

was given on the 9th in two doses, and after then 10 grs. daily. Patient was discharged well on the 17th, and when last seen two months later was well. The blood was examined on two occasions on the 10th May and nothing observed.

It will be seen that again, as was the case in March, we have clinically a quotidian fever due to a double generation of the tertian parasite. On each occasion in which the blood was examined on the 7th and 8th two different generations of parasites were observed. The history of the case is that of typical double tertian, the symptoms of which, looked at from a clinical point of view only, might legitimately be termed those of a quotidian fever. Microscopical examination, however, showed the simultaneous presence of the tertian parasite in two stages of development, the motile stage observed on a given day indicating the presence of parasites half developed and progressing to the sporulating stage, which would be coincident with a fresh attack of fever on the following day. It must be noted that coincidently with the presence of tertian parasites were found examples of the crescent form of malarial parasite. The exact clinical import of this form of parasite in the present case it is difficult to appreciate as the patient was not sufficiently long under observation, such clinical manifestations of disease as it was capable of producing being masked by the more pronounced symptoms due to the active tertian amœba. The association of the crescent form with the tertian parasite appears to be one of the commonest forms of mixed infection, and affords examples which can be used in support of the theory that one form of the parasite may be transmuted into another. Grassi and Feletti report three cases in which crescents only were found in the blood after a period during which the tertian parasite only had been found. That cases of this kind are, however, due to mixed infection, in which for a time the presence of one parasite is in such small numbers as to be masked by the prevailing form, has, it

seems to us, been clearly shown both by the observations of pure cases (single infection) and by the results of the artificial production of malarial fever by the injection of malarial blood.

We now give a case in which a typical double tertian was produced by two alternate generations of the tertian parasite uncomplicated by any other form, the case in this respect being typical of many others observed by Dr. Marshall in Spain during the spring and summer of 1895.

Case 4.—Manuel S. Sanchez, æt. 26, a strong young miner, has lived for six years in the district, and had fever for the first time a month ago. He took quinine and was then free from it for twenty-six days.

He states that he had attacks on June 17th, 18th, 19th, and 20th. Cold stage is severe, attack generally occuring at midday. There is an herpetic eruption on the lips. He states that the fever has been always quotidian.

June 20th.—When seen at 6 p.m. he was sweating, the attack passing off.

Microscopical appearances.—8 p.m. Parasites filling more than two thirds, with peripheral pigment.

21st.—8 a.m., 96·7° F.; 12 a.m., 104·5° F.; 4 p.m., 99·6° F.; 8 p.m., 100·6° F.; midnight 98° F. Cold stage began at 11 a.m. and was very severe.

Microscopical appearances in fresh and stained preparations.—8 a.m. *a.* Large parasites with scattered pigment. *b.* Large sterile forms. *c.* Forms filling one third to one half or more, pigmented. 10.30 a.m. *a.* Sporulating parasite. *b.* Parasite filling one half to one third, with pigment. *c.* Large sterile parasite. *d.* Segmenting form; several grains of pigment moving slightly. 4 p.m. *a.* Parasites filling one quarter to two thirds of red corpuscle. *b.* Small parasite in red corpuscle, spore size and unpigmented. *d.* Sterile forms. At 8 p.m. Quin. Sulph. 10 grs. given. The generation set free in to-day's attack had already attacked the red

corpuscles, and would, if not destroyed, have produced fever on the 23rd.

22nd, 12 noon.—Temp. 98·2°. Cold stage less severe than last; began at 4 p.m. Temp. 4 p.m., 99·6° F.; 6 p.m., 104·4° F.; 8 p.m., 101° F.; 12 midnight, 97·6° F. The attack was here delayed four hours by the quinine given. Quinine sulphate 10 grs. given morning and evening.

Microscopical appearances.—8 p.m. *a.* Parasites one third to one half of red corpuscle. *b.* Parasites filling one half to whole, with scattered pigment. *c.* Large sterile forms—some in red corpuscles which were much enlarged and discoloured, being represented by very faint outlines. 4.20 p.m.—*a.* Parasites filling one fourth. *b.* Sporulating forms. *c.* Sterile forms. *d.* One parasite half filling red corpuscle, pigmented. *e.* Large parasite with scattered pigment, with outline of decolourised red corpuscle.

23rd.—No fever. Quinine sulphate 10 grs. given morning and evening.

Microscopical appearances.—8.30 p.m. *a.* Parasite observed filling one quarter of red corpuscle.

24th.—9 a.m. Parasite observed filling one quarter. Quinine sulphate 10 grs. morning and evening. Discharged well on the 27th. On the 24th and 26th only pigment-bearing leucocytes and free pigment seen in the fresh blood.

In this case it will be seen that synchronously with a daily crisis sporulating parasites were present in the blood, accompanied by half-developed parasites which, if their growth went on unhindered, were ready to produce another paroxysm the following day. After the administration of three 10-grain doses of quinine the fever was arrested, and after that time undeveloped and sterile parasites were observed. A very slight rise in temperature (99·5°) on the 24th at midnight, was probably due a very few surviving parasites delayed in their development by the continued administration of quinine. When dis-

missed from hospital on 26th he was directed to continue to take quinine for some time, and the cure was complete.

The following case is quoted to illustrate how a certain number of parasites may be present in the blood without invariably producing a febrile attack.

Case 5.—Emenejelva Ameijeiras, æt. 39, has had fever on and off for a year, generally tertian. The cold stage is severe, and the hot stage is accompanied with severe headache. Says he has had fever for four days, but not at regular hours.

July 11th, 11.30 a.m.—Feels hot, but not very ill. Temp. 12 noon, 101·8° F.; 4 p.m., 101·2° F.; 8 p.m. 101·2° F.; midnight, 98° F. Profuse sweating at 8 p.m.

Microscopical appearances.—11.30. a.m. *a.* parasite filling one third of red corpuscle, with scattered pigment. *b.* One with slight peripheral pigment and numerous oval and irregularly shaped patches in red corpuscle stained blue (methylene blue staining). *c.* Sporulating body; spores separate. 4 p.m. *a.* Small number of parasites half developed. *b.* Pigment-bearing leucocytes.

12th.—Purge of Magnes. Sulph. No fever all day.

Microscopical appearances.—9.30 a.m. *a.* (In fresh blood), small parasites with very fine pigment. *b.* Segmented forms. *c.* Sterile form. 4.30 p.m., several half-sized parasites.

13th.—Fever came on at 9 a.m. with severe shivering, Temperature 8 a.m., 98·6° F.; 11.30, 104° F.; 4 p.m., 99·6° F.; 8 p.m., 99° F. Quinine sulphate, 15 grs., at 8 p.m., and on 14th at 8 a.m.

Microscopical appearances.—9 a.m. *a.* Sporulating bodies, one with sixteen spores. *b.* Young parasite. *c.* Pigment-bearing leucocytes. Under administration of quinine 15 grs. daily the fever was arrested.

It will be observed that although sporulating forms were found on the 12th, no febrile paroxysm occurred on that day. The generation due for that day had already been observed to be scanty on the 11th, and the man had

been freely purged on the morning of that day, two facts which must be borne in mind in connection with the absence of a paroxysm.

The Tertian Parasite, Hæmamœba Vivax (Grassi and Feletti).

This parasite, which undergoes development in twenty-four hours, is found in the early stage as a small, round, pale body adhering to the red corpuscle. Later it is found within the red corpuscle, which becomes progressively enlarged and paler with the growth of the parasite, till in the mature forms only a small rim of corpuscle is found, almost entirely deprived of hæmoglobin. The young parasite has a most active amœboid movement in the red corpuscle, throwing out arms in all directions and assuming the most fantastic shapes. As it enlarges it becomes less active, till we come to the mature sporulating body which is quite quiescent.

Pigment early shows itself in very fine granules, contrasting in this respect strongly with the coarser granules of the quartan. This pigment has a most active, swarming motion, and retains this property, though to a less extent, up to maturity, when it becomes still and congregates in the centre preparatory to sporulation, which takes place by the division into twelve to sixteen spores, that are smaller than those of the quartan.

The features distinguishing this parasite from the quartan are as follows:

The amœboid movement is more active than in the quartan, in which it is comparatively slow.

The pigment is finer at corresponding stages, and has a very lively movement.

The spores are small in size, but more numerous than those of the quartan.

The red corpuscle, which in the quartan is little, if at all, altered in size and colour, in this case becomes very

early enlarged and pale, and by not offering a sufficient contrast, renders the parasite more difficult to find in fresh preparations.

The sterile forms resemble those found in the quartan.

IRREGULAR FEVERS (INCLUDING TRUE QUOTIDIAN FEVERS).

We shall now give a few examples of the fevers which in the present condition of our knowledge may be provisionally and conveniently termed irregular fevers. They are frequently chronic, and are often obstinate, as the parasites are not so easily destroyed by quinine as in the tertians and quartans. Sporulation takes place in the internal organs, the blood from the finger seldom showing sporulating forms, the parasites which are found in it being mostly in an early stage of development, and often very few in number. It is in a certain proportion of these forms of fever that the crescent body is found, and in which extensive changes are produced in the spleen and liver by the development of the parasites in the tissues. Severe forms of fever—the types often termed "remittent,"—and forms of malarial cachexia without febrile attacks, are both due to the parasites of this class. These irregular fevers manifest themselves in the south of Spain in summer and autumn, but being exceedingly chronic and obstinate are found in patients at all seasons of the year. The cases found in winter and spring are those which had begun at the end of the previous summer, and have remained uncured. They correspond in this and in many other respects to the summer-autumn fevers of the Italian physicians. The following cases are examples of these irregular fevers as observed in the south of Spain.

CASE 6.—Francisco Nunez Rodriguez, æt. 42, a miner, admitted December 5th, 1894.

The patient, a weakly, thin man, with a very dirty sallow complexion, has suffered more or less from fever since August 27th. The type has varied, and quinine for a time cut it short, but it soon returned. For the last few days he has had daily attacks beginning in the afternoon. Three days before admission he took a purge and 6 grs. quinine daily for the three days. Lungs healthy.

December 5th.—10 a.m., temp. 98·4° F.; 12 noon, temp. 98·4° F. 2.30, pains in bones, and tenderness over spleen. 4 p.m., temp. 100·1° F.; has had slight sensation of cold. 8 p.m., temp. 102·8° F.; feels warm, less pain. Slight sickness during night, then sweating, and relief from all pain.

Microscopical appearances.—10 a.m. Parasite occupying one third with fine pigment points. Noon. *a.* Unformed masses with pigment. *b.* Small parasites filling one third to one sixth of red corpuscle. 4.10 p.m. *a.* Unformed masses free in the blood, containing small rounded clumps with pigment granules attached. *b.* Small free clump with pigment attached, apparently débris of a sporulating parasite.

6th.—9.30 a.m. Comfortable; temp. 99·8° F.; 12 noon, temp. 99·2° F.; 4 and 5 p.m., temp. 100·6° F. Slight shivering and headache in afternoon. 8 p.m., sweating, temp. 100° F.; midnight, temp. 98·4° F.

Microscopical appearances in stained preparations.—9 a.m. *a.* Parasite filling one half, with central comma-shaped pigment. *b.* Blue-stained body, filling nearly one half of red corpuscle. 12 a.m. Small spherical body with central pigment.

7th.—Temperature normal and subnormal till afternoon. 4 p.m. Temp., 98 8°F.; no cold or pain. 8 p.m. 100·6° F. Midnight the same. Sweating at night. 9 a.m. One gramme quinine sulphate in solution given.

Microscopical appearances.—9.30 a.m. Spherical agglomeration of pigment in fine granules in centre of red corpuscle, indicative of crescent formation. 4 p.m. *a.*

Ruptured red corpuscles. *b*. Crescent. *c*. Small round parasite.

8th.—9 a.m. One gramme quinine sulphate given. Temp. 4 a.m., 99° F.; 8 a.m., 99·4° F.; noon, 99° F.; 4 p.m., 101·4° F.; 8 p.m., 99·4° F.; midnight, 98·8° F. Slight attack preceded by headache and sensation of cold. Short hot stage which ended by sweating.

Microscopical appearances.—10 a.m. *a*. Crescent. *b*. Small round parasite. *c*. Parasite filling one fifth of red corpuscle with central pigment. 4.30 p.m. *a*. Crescent. *b*. Small parasite.

9th.—Temperature subnormal and normal. Headache and deafness. Sweating at night slight.

10th.—Condition the same as on the 9th. Temperature rose slightly in the afternoon. 9.30 a.m. *a*. Small spherical body in red corpuscle. *b*. Crescent. *c*. Small parasite.

13th.—Temperature normal.

Microscopical appearances.—9.45 a.m. *a*. Crescent. *b*. Red corpuscle containing spherical pigment in centre (outline of parasite not well defined). *c*. Small parasite.

14th.—Temperature normal.

Microscopical appearances.—9.45. *a*. Small, free, rounded clumps with pigment granules attached. *b*. Crescents and small parasites.

17th.—Discharged. Feeling well when seen some months after.

There is to be noted in this case that the parasites found in the blood from the finger were small, and in an early stage of development; that some parasites were found with the spherical central mass of pigment, which is distinctly indicative of crescent formation; that crescents were found free in the blood; then after rest in the hospital and administration of two doses of 15 grs. quinine, the temperature became normal, and the fever crises ceased, but the blood continued to show crescent forms. The patient was seen occasionally for four months afterwards, and appeared to be well. No

more fever attacks occurred, but there was no opportunity of examining the blood.

A point of interest in this case, as regards the microscopical appearances, is the fact that the parasites in the blood from the finger were found either in a condition of small bodies, or bodies filling half the red corpuscles containing pigment, or crescents.

The clinical symptoms and the temperature chart are those of a quotidian type of fever, and the forms of the young parasites found would not be inconsistent with those described as characteristic of the quotidian parasite by the Roman school. It would be quite in accordance with the teaching of that school to find crescents developing from the quotidian parasite. On the other hand, there is nothing in this case that is incompatible with the theory of Grassi and Feletti, that in instances like these we have to do with the crescent parasite as a separate species—genus *Laverania*, species *Laverania malariæ*, of these authors—and the young forms would be regarded throughout as early forms of the crescent body. It will be observed that no sporulating form was found in the blood from the finger.

Case 7.—Juan Garrida Martin, æt. 18, admitted December 20th, 1894. States that he has been suffering at intervals for four months from fever, and that on the 18th and on the 19th he had a paroxysm.

December 20th.—Temperature 1 p.m. 98·6° F.; 3 and 9 p.m., 98° F.; midnight, 101·4° F. Mild paroxysm.

Microscopical appearances in stained preparations.—3 p.m. Number of red corpuscles with round and horse-shoe-shaped pigment in a small clear space in the corpuscle. 4.30 p.m.—*a*. Parasite filling one half of the red corpuscle with central pigment. *b*. Parasite nearly filling red corpuscle with fine peripheral pigment. *c*. Parasite filling one quarter of the red corpuscle with nine minute hæmatoxylin-stained peripheral points in its circumference.

21st.—Temperature 9 a.m., 96·6° F.; 4 p.m., 99° F.;

8 p.m., 99° F.; midnight, 104·6° F. Rather severe paroxysm.

Microscopical appearances.—10 a.m. *a.* Spherical body with central point of pigment nearly filling red corpuscle. *b.* Round body with peripheral pigment filling two thirds of the red corpuscle. 2 p.m. *a.* Free oval body, smaller than a red corpuscle, with very fine pigment. 4 p.m. Minute spherical bodies in red corpuscles with central point.

22nd.—4 a.m. Temp. 100·6° F.; 8 a.m. to midnight, temperature 98° F. No paroxysm this day.

Microscopical appearances.—10 a.m. *a.* Hæmatoxylin-stained body filling three quarters of a red corpuscle with two more deeply stained points in the periphery. *b.* Many minute pigment points in clear spaces in red corpuscles. 2 p.m. *a.* Two parasites occupying three fifths of a red corpuscle, apparently joined in the centre. *b.* Hæmatoxylin-stained body nearly filling red corpuscle (? dead parasite). 7 p.m. *a.* Many minute pigment points in clear spaces in red corpuscles. *b.* Great variety of small stained bodies with small hæmatoxylin-stained points. *c.* Hæmatoxylin-stained body filling two thirds of a large red corpuscle, with scattered pigment. *d.* Not well defined appearance of sporulation. *e.* Small parasites filling one fifth to one quarter of the red corpuscle. *f.* Small parasites on the periphery of the red corpuscles.

23rd.—Temperature 4 a.m., 102·8° F.; 10 a.m., 98·4° F.; normal for the rest of the day. Slight paroxysm.

Microscopical appearances.—10.30 a.m. *a.* Small parasite. *b.* Parasite filling one fourth to one third of the red corpuscle with central pigment.

The patient here passed out of observation till December 29th, owing to the absence of Dr. Marshall.

On the 24th there was a double paroxysm with a distinct intermission for about an hour.

25th.—Short paroxysm with high temperature—104·6° 9 a.m.; temp. 98° F. at 4 p.m.; 11.30, 99·6° F.

26th.—Temp. 101·3° at 4 a.m.; 99·2° F. at 8 a.m. and 4 p.m.; 98° F. at midnight.

27th.—Severe paroxysm; temperature reaching 104·4° at midday. Ten grs. quinine given at 9 p.m.

28th.—Temp. 98° F. at 9 a.m.; 10 grs. quinine sulph. given. Very severe paroxysm. Temp. 105·6° at 4 p.m.; 10 grs. quinine given in the evening.

29th. — *Microscopical appearances.* — A parasite was found filling one third of red corpuscle.

The temperature remained from this time normal, and he was discharged on January 2nd apparently well.

A glance at the Chart in this case, with a consideration of the symptoms, will show that it answers to the description of quotidian fever as given in Marchiafava and Bignami's work. The parasite as we have described it corresponds sufficiently accurately to the quotidian parasite of these authors, the *Hæmamœba præcox* of Grassi and Feletti. It will be observed that although the blood was examined twice, and sometimes three times daily from the 20th to the 23rd, and again on the 29th, no crescent forms were ever found. Nor can it be said that this was due to the short duration of the fever. According to the man's own statement he had paroxysms two days preceding his admission, and he had a daily paroxysm in the hospital until the 28th of December, so that although there were at least ten days' fever the parasites did not develop into the crescent form. The Roman school would not consider that to be inconsistent with their doctrine, for Marchiafava and Bignami state that there are a considerable number of cases in which the crescents do not appear. On the other hand Grassi and Feletti would explain this case by considering the parasite to be of the pure quotidian variety without any admixture of the crescent species, and that the reason no crescent forms were found was because that variety was not present in the blood, the quotidian never being transformed into that species. In this case there is to be noted the irregularity of the crisis, the number of small parasites,

and the comparative absence of sporulating forms in the blood from the finger, only one being found.

The condition of the pigment in the parasite in this case is characteristic and deserving of notice. In many instances its presence in the red corpuscles was shown by a small colourless sphere which contained in its centre a minute comma or horse-shoe shaped mass of homogeneous-looking pigment without any appearance of sporulation, contrasting in this respect not only with the arrangement of pigment in the quartan and tertian parasite, but also in that of some other cases of irregular fever. One of us has found similar appearances in the blood of cases which have occurred in the West Coast of Africa, and this peculiar arrangement of pigment would seem to have a certain diagnostic bearing.

There are cases of long-continued chronic fever in which the patient feels out of health, but in which the rises of temperature are small and infrequent. The crescent body is not always found in these cases in blood taken from the finger, and it is not always easy to determine from the parasites which are found in the blood to what variety they belong. The following case is given as an example of the difficulty that may exist for a time in establishing the final diagnosis. No crescent forms were found, and only one form resembling a sporulating body with central pigment.

Case 8.—Josè Sanchez Concession, æt. 24, admitted April 18th, 1895.

Patient states that he has suffered at intervals for a year from malarial fever of tertian type, that the present attack dates from the 11th, and that paroxysms occurred on the 13th, 15th, and 17th.

He is weak looking and cachetic. Spleen enlarged. During his stay in the hospital he had no attack. The temperature rose for an hour or two daily to 99° F. On the 24th and 28th it rose to 100·2° F. without a cold stage (see Temperature Chart). On the 27th he com-

plained of pain in the spleen, for which an ointment of belladonna and hydrarg. was ordered. Discharged on May 7th well and stronger.

No quinine was given or taken for some time before admission. Tonics were given.

Microscopical appearances.—April 18th.—10.30 a.m. *a.* Several parasites filling more than two thirds with peripheral pigment. *b.* One filling more than one half; no pigment. *c.* Parasite filling one third in enlarged pale red corpuscle. *d.* One segmented body. 3.30 p.m. *a.* Large parasite with scattered pigment. *b.* Small parasite filling one third with pigment. *c.* Free small parasite with filaments (flagella).

April 19th.—10.30 a.m. *a.* Small spherical body with peripheral pigment points. *b.* Parasite filling one third, pigmented. *c.* Large parasite filling red corpuscle, with scattered pigment. 3 p.m. *a.* Parasite filling one third to one half. *b.* Several parasites with very little pigment.

20th.—8.30 a.m. *a.* Parasite filling one third to one half or more with very fine, scattered pigment. *b.* Small parasite.

21st.—11 a.m. *a.* Stained parasite filling one third of red corpuscle. *b.* Irregular shapes one quarter to one third with fine peripheral pigment.

22nd.—9 a.m. Same as above.

24th.—9.30 a.m. *a.* Parasite nearly filling red corpuscle with two minute dots of pigment. *b.* Red corpuscle two thirds filled with bluish-stained body (hæmatoxylin); no pigment. *c.* Three stained small spherical bodies in a red corpuscle touching each other.

26th.—10 a.m. *a.* Parasite filling from one third to one half; no pigment.

29th.—3 p.m. Several parasites filling one quarter to one third or more, pigmented.

30th.—8.30. Three sterile bodies.

We give another similar case because a crescent body was found with parasites which resemble the early forms of the tertian parasite.

Case 9.—Manuel Trigo Fernandez, æt. 16, admitted June 7th, 1895. States he has had fever for five months, on and off, of a tertian type. He has taken quinine during that time, but none for the last week. He had attacks on the 2nd, 4th, and 6th, with severe pain in the head and slight cold stage, but no stage well marked. Two years ago he had tertian fever for seven months.

June 8th.—Temp., 8 a.m., 98° F.; 12 noon, 99·8° F.; 4 p.m., 100·4° F.; 8 p.m., 98·2° F.; 11 a.m., pains in limbs and head; attack mild.

Microscopical appearances.—7th.—10 a.m. (Stained preparations.) *a.* Small circlet with more deeply stained, small, rounded body in the ring, on the border of a red corpuscle. *b.* Clear space containing hæmalum-stained body filling two thirds of a red corpuscle. *c.* Parasite filling one half. *d.* Small parasite filling one fourth, pigmented. *e.* Large parasite filling two thirds with scattered pigment. 4 p.m. *a.* Red corpuscle filled with large sterile parasite (scattered pigment). *b.* Crescent. *c.* Red corpuscles with pigmented parasites filling one third to one half. *d.* Sterile forms.

8th.—8 a.m. *a.* One small parasite in enlarged red corpuscle with slight pigment. *b.* Large red corpuscles with parasites filling one half or more, with peripheral pigment. *c.* Sterile forms. 3 p.m. *a.* Small unpigmented parasite in red corpuscle. *b.* Large forms filling three quarters to whole. *c.* Sterile forms.

9th.—Temp. 99·6° F. at 8 a.m.; normal for rest of day.

10th.—Temp. 99·4° till 8 p.m. (thereafter normal till discharge).

Microscopical appearances.—8.45 p.m. One large parasite occupying three quarters of red corpuscle.

The case was very mild. The boy was well nourished and had no enlargement of the spleen. He had slight anæmia. Fifteen grains quinine given on the morning of the 10th. Ten grains of quinine in two doses daily on 11th and 12th, on which day he was discharged.

It is to be noted that in this case sporulating forms in

the blood from the finger were not found. The case was not well marked, the temperature was not high, and a crescent form was found, indicating that the patient was the subject of chronic malaria. What relation the presence of the crescent body had to the febrile attacks it is difficult to say, but it had an undoubted relation to the anæmic condition. Possibly a more exhaustive examination of blood from the finger might have resulted in the discovery of a sporulating form.

Now that it has been definitely established that malarial fever is due to a hæmatozoon, the question as to how it gets introduced into the human body becomes very prominent. Various factors have been long known to be concerned in the production of malaria, particularly certain conjoint conditions of temperature and moisture. These conditions we now recognise must owe their power to the influence which they have on the development of the hæmatozoon, but how the parasite gets introduced into its host is yet unknown. It becomes interesting, therefore, to examine carefully once more all the conditions that are known to favour the development of malaria, with special reference to this problem. For this reason we shall notice some of the conditions that are associated with malarial fever in the south of Spain, our experience being limited to that part of the Province of Huelva in which the observations were made.

Soil.

The aspect of the country is that of a series of short hills, with intervening ravines or valleys, in many of which there are streams in winter. These dry up in the summer, leaving pools. There are no marshes. It is about 320 mètres above sea level. The soil is firm, and water does not lie on it long. There is no vegetation on account of sulphur smoke, but about twenty years ago it was largely wooded, and rich in orchards.

Some of the villages are built on the face of a small hill, with a watercourse at the bottom. The houses inhabited by workmen have, for the most part, earthern floors, or stones or tiles placed on the earth. The streets in the villages are unpaved, or have only stones set into the ground. The houses in the town of Rio Tinto have floors of flat tiles.

The houses in village A have wooden floors on the ground floor. Here the men for the most part sleep on the first floor. In the town they sleep on the ground floor.

In the surrounding villages the houses are one-storied.

It is interesting to observe that here, as in other parts of the world, the malaria is often found to be strictly localised, a very short distance being often sufficient to divide a fever-stricken from a comparatively fever-free situation. Although we have not been able to discover differences between fever-free and fever-producing localities which are near each other, it may be useful to emphasise the fact that these differences exist by relating what has come under our own notice.

Localisation.

It is well recognised in the district that there are some spots where malaria is always prevalent in the fever season, and others, as for example the town, where it is not found to any great extent. In fact people in the town run little danger of catching the fever, while people living in village A, half a mile off, even although they sleep on the first floor, are more liable to be attacked. For example, an Englishman who always had fever when living in village A, when he removed to the town was seldom affected. Another man who lived six years in the town without catching fever, went to live at village A and was attacked. To illustrate how narrowly the infection may be localised we may refer to two villages which are distant from one another about a quarter of a mile. B is on a plateau; C lower down on the other side of a stream, and on the

side of a hill. C is notoriously malarial; B is much more healthy, although the houses are similar in each.

About 300 yards from the village A, there are houses on a small elevation with a watershed on each side, inhabited by foreign workmen. Here fever is very common, and three people out of a group of eight have been affected with fever at one time.

Most of the malarial villages are situated on the face of a hill, with a stream, which is more or less dried up in summer, at its foot. In the same village there are streets and even houses where it is more frequently met with than at other places. Sometimes in a house liable to the infection Dr. Marshall has seen four patients suffering simultaneously from fever, and it is not at all uncommon to find more than one patient down with it at the same time in the same house or same room.

Facts such as these would seem to indicate that the parasite, in the stage in which it gets admission into the circulation, not only hugs the soil, but that it is not readily carried by currents of air from one part to another, and we may reasonably suppose from the facts observed here (as elsewhere), that moist conditions possibly produce some change in the parasite in its extra-corporeal life, and that those changes render it more fitted to establish itself in the human body.

Here, as in other countries, change of locality often has an excellent result in curing the fever. For instance, a Spanish servant was ill in one street with fever, and when removed to another house she almost at once lost it. Two Englishmen at Punta Umbria, with a slight form of fever every day, ceased to have attacks from the day they were removed to Huelva Hospital. Such recoveries are too rapid to be entirely dependent on removal from sources of infection, and must be largely attributed to the tonic effect of change.

Disturbance of soil is a well-known cause of activity in the infection by the malarial parasite, and this has been observed in this district. During certain seasons in

which there had been extensive foundations excavated for dams and houses, and when the houses in certain villages were first built, there was much more fever than generally has occurred in them since. One Englishman was seriously attacked with fever while superintending the making of a tennis-court in front of his own house for which much soil had to be removed.

We can only account for the disturbance of the soil producing fever by presuming that it sets free into the air a number of malaria parasites in a condition ready for beginning their intra-corporeal life, and although the many researches which have been made with the object of detecting the parasite in the soil have not, so far, been conclusive, it is evident that this is a line of research which will not be abandoned until the problem has been solved.

Drinking-water has often been suspected of being the means by which the parasite is admitted into the body. Hitherto this has not only not been proved but there are many facts which render it improbable. The hypothesis is certainly not borne out by what has been observed in the district where our cases were observed.

The drinking-water in this district is pumped out of wells. All the English people are supplied from the same wells, although living in different parts of the district; yet those persons who live in the town are remarkably free from fever, whilst those in village A and those living on the elevation 300 yards from it, before alluded to, are more liable to the infection.

The two villages B and C, contrasted before, get water indiscriminately from the same two wells, yet one is more seriously affected with fever than the other.

So far there is no sufficient evidence that the parasite gets into the system by the mouth. The experiments of the Roman School of Hygiene are against water containing the infection (Grassi and Feletti); pigeons which were made to swallow dew collected in malarial districts remained immune. Calandruccio and Grassi drinking

from 30 to 50 grammes of dew also remained immune, as did Calandruccio after swallowing considerable quantities of mud which might well have been considered to contain the malarial poison. Nay, even malarial blood may be swallowed both by human beings and pigeons without causing malaria. (Pigeons and some other birds are affected in malarial districts with a hæmatozoon similar to the hæmatozoon of malaria in man).

Laveran, considering that mosquitoes are found in malarial districts, and that draining the soil, which prevents fever, also destroys mosquitoes, suggests the possibility of these insects acting the part of an intermediary host for the malarial parasite, as they do for the filaria. Grassi and Feletti object to this hypothesis from the fact that birds which become infected with hæmatozoa in malarial districts are not attacked by mosquitoes. A pigeon, in which the blood is ascertained to be quite healthy, when suspended at a certain distance from the ground over a malarial soil, becomes the subject of a hæmatozoon which if not identical with that of the hæmatozoon which attacks man, is allied to it, and mosquitoes and insects can hardly, in this case, be held responsible for the infection. Further, Calandruccio, who has investigated the subject, states that the malarial parasite dies in the intestine of the mosquito without undergoing any further development, the same fact being verified for the leech. It is further well known that, as Grassi and Feletti remark, many places are full of mosquitoes without having malaria. Malaria exists in Sicily in places where there are no mosquitoes that bite. (Oral communication by Prof. Feletti.) It was stated by Dr. Manson, at the annual meeting of the British Medical Association in 1895, that Surgeon-Major Ross, working in India, had found that the parasites are able to resist destruction in the stomach of the mosquito, and considers that a development in this insect as an intermediate host is evidenced by the fact that a flagellate form is more frequently found in blood which has been in the mosquito than

in blood from the finger, and that the crescent forms all become spheres. A full account of the paper in which this statement is included has not yet been published, but so far as these two statements go they are not convincing. The right appreciation of phenomena of this kind requires the special knowledge of the lower forms of life which is possessed by zoologists. On such a point, therefore, the association of Professor Grassi in the memoir bearing his name with that of Professor Feletti deserves much consideration.

These authors remark that the assumption of the rounded form may indicate approaching death in many protozoa (Mastigofori, Amœba, &c.), and that at the same time that the crescents throw out flagella they throw out round hyaline bodies, three, four, or more, of different sizes (the cytoplasma of the parasite), similar bodies being thrown out by many protozoa. In comparison with the setting free of flagella by the flagellata, they state that in the case of a crescent body the separation of the flagellum follows too soon on its production. They quote instances in spermatozoa which undergo similar changes and movements in the death agony.

From the facts which they adduce they conclude that certain forms of life, and certain organised elements when placed in abnormal media, react by unusual and irregular movements, which usually precede death. The crescent forms in blood removed from the circulation thus find themselves, from the blood coagulating, in an abnormal medium, and it is not to be wondered at that they change into flagellated bodies before dying.

These are points on which we do not consider that our opinion is entitled to weight. We do not lay claim to special zoological knowledge, and we have not applied ourselves to the study of this particular question. The subject is, however, so important, that our paper would be incomplete without a reference to it. The instance of the man cited by Surgeon-Major Ross, who, eleven days after swallowing a bottle of water containing malarial

mosquitoes, developed malarial disease, is on the other hand, of importance. As a solitary instance it cannot claim great weight, but if confirmed by similar examples it would prove to be a fact of much value, as indicating that in addition to the ordinary exogenous development of the malarial parasite, which undoubtedly exists, the hæmatozoon could also be conveyed through water by the mosquito.

The mosquito theory does not find much support in the district we are considering. Mosquitoes are indeed found, but not in numbers sufficient to be unpleasant, mosquito nets not being necessary. Flies are more common, and many of them bite.

The sulphur smoke and consequent want of vegetation account for the comparative absence of insect life, although they do not seem to have any specially injurious effect on the extra-corporeal phase of life of the parasite of malaria.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. viii, p. 125.)

DESCRIPTION OF PLATE I,

The Parasite of Malaria (Robert J. Marshall, M.B., C.M., and George Thin, M.D.).

Figs. 1 to 14 represent the quartan parasite from its earliest stage after attacking the corpuscle to its full development by the production of a new generation of spores.

Figs. 15 and 16 represent free pigmented bodies in the blood, which we consider as sterile forms of the quartan parasite.

Fig. 17 represents small pigmented bodies with pigment granules found free in the blood in quartan fever.

(These drawings were made by Dr. Marshall from preparations of fresh blood.)

Figs. 18 to 29 represent the progressive development of the tertian parasite from its first attacking the red corpuscle to its complete development into free separating spores.

Figs. 30 and 31 represent free pigmented bodies of the tertian parasite, which we consider to be sterile forms.

Fig. 32 represents pigmented bodies relating to the tertian parasite of various sizes found free in the blood. In one of these a long flagellated process is seen with swellings.

(These preparations were all drawn from the fresh blood, except Figure 28, which was drawn from a dried and stained preparation from the same patient as the others.)

Figs. 33 to 48 represent the appearances presented by the parasite in a case of irregular fever. Many crescents were found in the blood. No sporulating forms were found. Transition forms from the small colourless amœba to appearances indicative of the crescent form are already evident in Figures 38 and 39.

(They were all drawn from the fresh blood, except Figure 48, which was drawn from a stained preparation to fill up a gap in the forms observed in the fresh blood. This is one of the cases which have not been described in the text.)

Figs. 49 to 63 represent the parasite as seen in a case of irregular fever in which no crescents were observed.

Figs. 49 to 53 represent appearances seen in blood preserved and examined in Pacini's fluid, the parasites being represented by a colourless space in which the dark pigment is shown very distinctly.

Fig. 54 represents a small pigmented mass seen in blood from the same case preserved and examined in Pacini's fluid.

Figs. 55 to 63 represent parasites from the same case in dried and stained blood preparations, the staining being effected by various hæmatoxylin methods.

Figs. 56 to 60 show distinctly stained nuclear or nucleoliform elements.

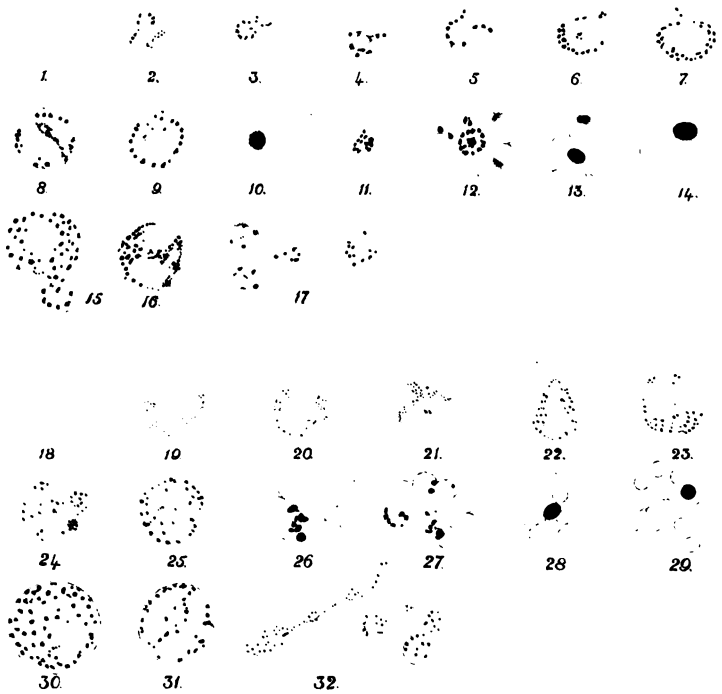
Fig. 61 shows stained thickened points in the periphery of the parasite.

Fig. 62 shows a parasite with very fine pigment scattered through it.

Fig. 63 represents a sporulating form. This was the only sporulating form observed in a very large number of preparations which were very carefully examined.

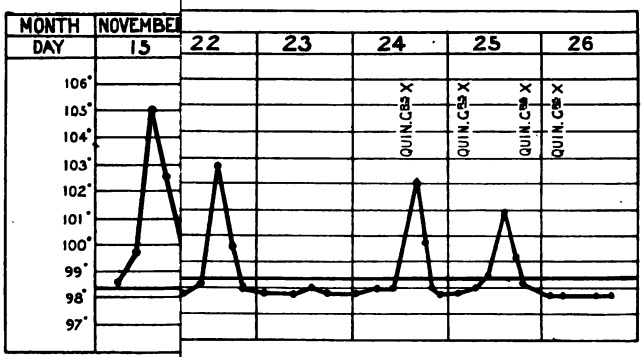
Figs. 64 and 65 represent circular bodies with stained points in blood from the same patient. They are drawn on a much more highly magnified scale than the other drawings in order to bring out very clearly the stained points. Their actual size was not a third of a red corpuscle. Similar small bodies with stained points have been described by other authors. They are quite distinct in appearance as well as in size from the ordinary sporulating forms.

It will be observed that in this case the forms of the parasite seen nearly all represented very early stages, Figures 62 and 63 being quite exceptional, showing that only the very young forms were to be found in the peripheral circulation.

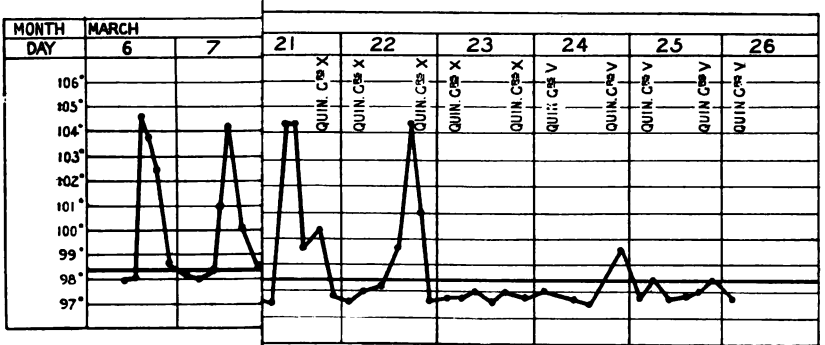

1.
2.
3.
4.
5
6
7
8
9
10.
11.
12.
13.
14.
15
16.
17
18
19
20.
21.
22.
23.
24
25.
26
27.
28
29.
30.
31.
32.
33.
34.
35.
36.
37
38
39
40.
41.
42.
43.
44.
45.
46.
47.
48.
49
50.
51.
52.
53.
54.
55.
56.
57.
58.
59.
60.
61.
62
63.
64.
65.

Danie

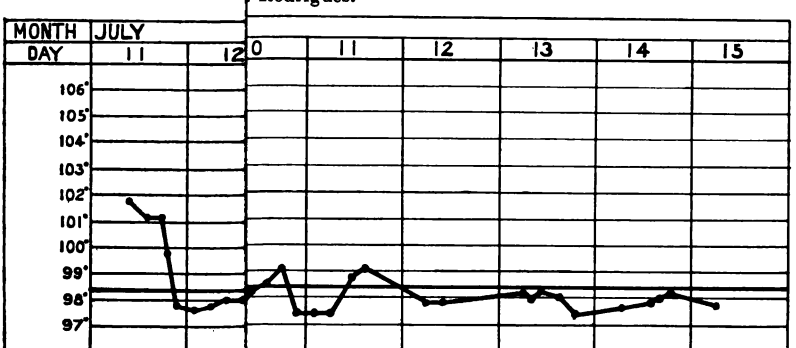
ɜE 2.—Josè Pantiga.



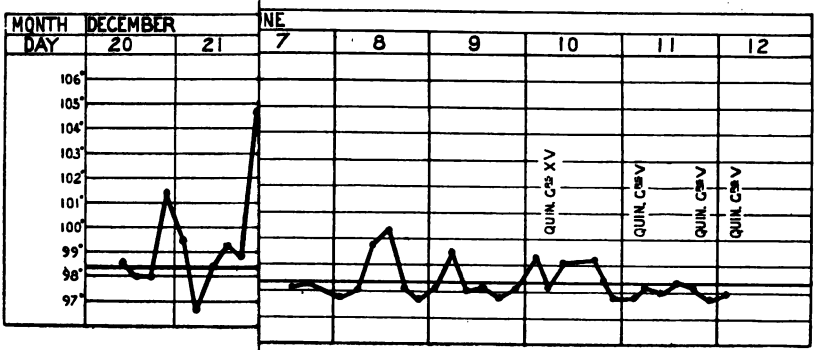
CASE 4.—M. S. Sanchez.

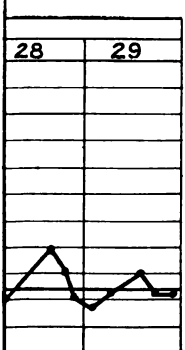


; Rodrigues.



CASE 9.—Manuel Trigo Fernandez.





A NOTE

ON THE

APPEARANCES FOUND IN THE TISSUES

IN A

FATAL CASE OF PERNICIOUS MALARIA AT SIERRA LEONE

BY

GEORGE THIN, M.D.

Received February 21st—Read February 25th, 1896

A WHITE man was admitted to the Hospital at Sierra Leone on October 11th, 1895, apparently suffering from alcoholism. His temperature at the time of admission was 100° F. He became delirious during the night, and although he understood questions which were put to him, he was unable to answer intelligibly. At 2 a.m. the patient was comatose, with a temperature of 106·2°. On the 12th he was very weak, and semi-comatose; temperature 98·4°. On the 13th he was comatose, and the temperature, which had again risen to 106°, was brought down by treatment to 102·4°. He died on the morning of the 15th. After death the liver was found to be much enlarged. The spleen was almost diffluent, and very dark. The surface of the brain was covered with lymph. The kidneys were slightly enlarged and congested.

Surgeon-Captain Duggan obtained the information referred to above from the medical man in charge of the patient, and was present at the autopsy. I am indebted to him for sending me portions of the tissues hardened and preserved in alcohol. I have not been able to obtain more detailed information regarding the case.

On examining these tissues I find the following appearances:

Brain.—The veins of the pia mater were found full of blood, and in some parts of them an unusual number of white corpuscles were mixed with the red corpuscles, but in the specimens I have examined I did not find parasites in these veins. Sections through the cortex, perpendicular to the surface, did not show any marked alteration in the brain tissue, with the exception of a number of oval and rounded colloid-looking masses scattered irregularly through the substance. The smallest of them were about the size of the nuclei of the ganglion cells; the largest were four or five times the diameter of these. This colloid-looking material was structureless, and stained with methylene blue and hæmatoxylin. Dr. Galloway has pointed out to me that similar appearances in the tissues of the brain and spinal cord have been described by pathologists, who consider them due to the action of the alcohol in which they have been preserved, and to have no relation to any particular disease. On my first examinations, the proximity of some of them to the sheath of the blood-vessels induced me to suppose that they were due to some inflammatory product which began to be deposited between the perivascular sheath and the brain substance, but subsequent and more prolonged examination showed me that this view was erroneous. The first indication of their being formed was seen to be a rarefaction of the fibrils of the brain substance, with slight uniform staining in the altered area. In the next stage, these fibrils had almost entirely disappeared, but the remains of a few of them could be detected in the coloured substance which was formed. The inference is

that they are the products of post-mortem change. In the specimens I examined they were situated at the junction of the white and grey substance, and were continued to a slight distance into the white substance. In one section, near the surface, over a small microscopic area, there was extravasation of red corpuscles into the brain tissue.

The capillary blood-vessels were found to contain many parasites. Some parts of the brain being better preserved than others, the relation of the parasite to the red corpuscle was only well seen in a certain number of sections, but successful sections were sufficiently numerous to enable this relationship to be accurately determined. In many of the smallest capillaries, the lumen of the vessel was filled with red corpuscles, and each corpuscle contained one, or in some cases two parasites. In the smaller veins a considerable number of red corpuscles were found without parasites, but there were still a considerable number infected, and those that contained parasites were generally found lying on the wall of the vessel. In some of the larger veins scarcely a parasite could be detected, but this does not apply universally. For example, in a vein of considerable calibre, the section of which, drawn to scale, is illustrated in Fig. 5, a very large number of parasites were found scattered amongst the red corpuscles.

In referring to the condition in which the parasites were found, it should be borne in mind that they change so rapidly after the death of the patient, that it is only when the autopsy has been made very soon after death, and the tissues have been most carefully hardened, that they are found in perfectly satisfactory condition. I do not know how long after death the post-mortem examination in this case was made.

The parasites were found in every stage of their development, from free spores which had just come into contact with the surface of the corpuscle to the stage of development in which a mass of central pigment was surrounded by a faintly-stained substance, which nearly

filled the spirit-shrunken red corpuscle. I have no doubt that if the condition of preservation had permitted, I should have been able to have coloured nuclear elements in the faintly-stained substance surrounding the pigment.

Not only were all the phases of development found in the brain, but frequently several stages could be found in the same vessel. Three such phases could be easily distinguished, namely, the stage with central pigment, which was comparatively rare, a smaller stage about half the size of the shrunken red corpuscle in which no pigment had formed, and a yet smaller stage seen as a somewhat minute, spherical, deeply stained body, occupying only a small part of the red corpuscle. This latter stage is by far the most prevalent in this case, and may be taken as indicating the prevalent stage of the parasite, not only as regards the brain but as regards the other parts of the body, in which it was, however, found in very small numbers. The larger phases were only observed in the brain. Many capillaries contained enormous numbers in that stage exclusively. A still smaller stage was observed exceptionally in the section of the vein to which I have already referred. The parasite in this case was seen in stained sections as a very minute round body, when looked at with ordinary powers appearing like a simple point, from which it appears that it begins to increase in size very soon after attacking the red corpuscle. In this section the number of red corpuscles which were attacked by more than one parasite was much larger than the proportionate number in the capillaries, in which most of the corpuscles harboured one, and only sometimes two parasites. As other large veins contained few or no parasites, it is evident that a great number of sporulating parasites in an adjacent capillary had simultaneously discharged their spores into the vein at this part, probably shortly before the death of the patient, when the circulation was becoming slow. In some sections deeply stained in Loeffler's methylene blue, a very delicate, faintly-coloured substance could be seen surrounding the more deeply

stained small sphere. Many of the capillaries in the white substance were empty, the chief manifest seat of the parasitic infection being the grey matter.

Very few white corpuscles were observed in the blood-vessels of the infected area of the brain. A few were found in the veins, in which occasionally, although as I have already stated very rarely, a large white corpuscle (the phagocyte of authors) could be seen in the midst of the uninfected red corpuscles, charged with spheres of pigment. On the other hand, many of the endothelial cells of the blood-vessels contained pigment, and in transverse sections of the capillaries it could be seen that the endothelial cell not only contained pigment, but was swollen and changed. Fig. 4 illustrates a transverse section in which the knife had passed through one such endothelial cell, which nearly occludes the lumen of the vessel. Although the obstruction to the circulation must be largely due to the capillaries being blocked with infected red corpuscles, yet the condition which I have now described, and which is represented in Fig. 4, must tend to increase the blood stasis.

Spleen.—The spleen was dark and soft, and except by the paraffin method, satisfactory sections could not have been obtained. The examination of stained sections showed that the only parts of the organ which were materially infected were the pulp. The Malphigian follicles were apparently unchanged. The nuclei of their cells stained very faintly. They contained, as a rule, very little pigment, or none at all, and such pigment as was observed was in the form of very small granules, whose arrangement corresponded to the capillary endothelium, and there is no doubt that the only pigment in the follicles was that which was contained in these endothelial cells. Practically, with this exception, they may be said to have been free from pigment. The connective tissue of the spleen did not seem to be changed.

In the stroma of the organ two distinct classes of cells could be distinguished. There were, first, are

the cells were mostly or entirely the recognised lymphoid cells, in which the comparatively large, round nucleus was surrounded by a small amount of protoplasm. The other cells were much larger and were of unequal size. Their shape was oblong or ovoid; and the protoplasmic substance, which stained deeply with eosin, was considerable even in proportion to the large rounded nucleus. These cells, though scattered freely through the stroma, were most numerous in the vascular parts of the pulp. They contained considerable quantities of pigment, dark in colour and spherical in form. In addition to the pigment the larger ones contained small colourless spheres, many of which had a central point of pigment which probably corresponded to decolorised, parasite-bearing, red corpuscles, such as have been described by the Italian physicians as being freely taken up by phagocyte white corpuscles. Many of the larger of these phagocytes contained vacuoles, and were evidently being broken up, and in some of them the process of disintegration had begun. The nucleus stained badly, in some instances scarcely at all, and the cell was evidently undergoing a process of necrosis.

A very little free pigment was observed in some sections, but nearly all the pigment seen was in cells.

The veins of the pulp contained many white corpuscles, some of them normal in appearance, and containing no pigment, but many were large, and contained spheres of pigment. Some of those which contained pigment were beginning to break down.

Very few parasites were found in the spleen. Very rarely a red corpuscle was observed to contain a small round parasite in an early stage, and in one section three red blood-corpuscles containing such parasites were seen juxtaposed, and lying on the wall of the vein.

Looked at as a whole the number of pigment-bearing leucocytes in the spleen was very large, and it could sometimes be observed that they tended to accumulate

around the Malpighian follicle, but did not enter it, a fact on which I shall comment later on.

Bone marrow.—A portion of rib which was sent me was broken up, and some of the pulp hardened and cut. No parasites were found in the sections examined. White corpuscles containing pigment were seen, but were much fewer in number than in the spleen.

Liver.—The most striking feature observed in sections of the liver was the quantity of pigment distributed between the rows of liver-cells. There were no signs of active inflammatory action in the liver substance. The hepatic cells were mostly apparently healthy and well-preserved, with the exception of a small area surrounding the central vein of the lobule. Round this central area the cells had undergone degeneration, being more or less broken up, vacuolated, and many of them completely destroyed. The destruction was most complete in the cells immediately surrounding the branch of the hepatic vein, but this central area of necrotic liver-cells was very small in comparison to the apparently healthy areas of the rest of the lobule, and it did not, even when most marked, amount to one fifth of that of the lobule. The capillary vessels between the rows of liver-cells contained many white corpuscles laden with pigment spheres and masses of various sizes, and the endothelial cells of the capillaries also contained pigment. Pigment-carrying white corpuscles could also be observed between the capillary vessels and the liver-cells, lying close to and in contact with the latter, their form being elongated in correspondence with the direction of the liver-cells. The pigment-bearing cells extended in rows from the periphery to the centre of the lobule, and were considerably fewer in number in the inner third of the area. Their number was sufficient, taken in conjunction with the swollen endothelium of the capillaries, to considerably obstruct the circulation in the lobule; and the destruction of the central hepatic cells, while the outer cells were healthy, may be considered due to the obstruction

of the blood-supply by the pigmented cells. In some sections, in certain areas the liver-cells contained small spheres of pigment, but these areas of pigmented hepatic cells were much smaller than the large areas in which the hepatic cells contained no pigment.

Kidney.—In the portion of tissue sent to me it was evident to the naked eye that some parts looked more congested than others, and in these reddened and congested looking parts the changes observed in the convoluted tubules were more extensive than in the parts which did not appear congested. The only structures in the kidney which were diseased were the convoluted tubules, and the changes in the epithelium of these tubules existed in all degrees—from swelling, granular degeneration of the substance of the cell, irregular broken-down contour, and loss of capacity on the part of the nucleus to stain, to complete breaking down of the cells. In the transverse sections of some of these tubules almost every cell was completely broken down and had disappeared, the only nuclei which were left being those of the cells of the basement membrane. The straight tubules were healthy. In the absence of parasites the only explanation of this acute necrosis of the secreting epithelium is to attribute it, as Bignami has done in the Italian cases, to the toxic substances produced by the sporulating parasites and excreted by the kidney. We know that the toxicity to animals of the urine is greatly increased in malarial fever.

The glomeruli were normal in appearance. A few isolated, minute groups of very small pigment spheres and granules were evidently contained in the endothelium of the capillaries.

There was no small-cell infiltration surrounding the blood-vessels or changes in the connective tissue which would indicate any interstitial inflammation. In some parts the blood-vessels were turgid with blood.

Very few parasites were found in the blood-vessels of the kidney : only very rarely, in a vessel a minute, stained,

round sphere in a red corpuscle indicated its infection by a parasite.

As, so far as I am aware, this is the first case in which the appearances observed by microscopical examination of the tissues affected with the malarial parasite of the pernicious fevers of the West Coast of Africa have been recorded, it is important to compare them with the description given by the Italian physicians of the pathological changes caused by the pernicious fevers of Southern Italy, and, if the comparison is made, the similarity of the changes in the tissues and in the appearance of the parasite in both cases will be at once apparent. It has been accepted by the Italian authorities that the pernicious fevers of Italy are caused by the parasite described by Marchiafava and Celli in the so-called summer-autumn fevers. This parasite and the changes it produces have been described in great detail by Dr. Bignami in a memoir entitled "Ricerche Sull' Anatomia Patologica Delle Perniciose" ('Atti della R. Accademia Medica di Roma,' 1890), in which thirteen cases are minutely described. It was shown by the observations made in these cases that in pernicious fevers the brain is usually found so full of parasites, that in some areas it is impossible to find a red corpuscle presenting a normal appearance. There is a complete injection of parasites, which is usually most manifest in the rich capillary network of the grey substance. The arterioles and small veins have fewer parasites than the capillaries. Generally, various phases of the cycle of the parasite are found, but one mostly prevails. Only in rare cases were capillaries found blocked with white corpuscles laden with pigment, the phagocytic action being mostly performed by the endothelium of the vessels. The pigment-carrying and altered endothelium sometimes obliterates the lumen of the vessels. Hæmorrhage was occasionally found in the perivascular sheath. The spleen was mostly characterised by the presence of multitudes of phagocyte white corpuscles which contained

pigment, parasites, and red corpuscles enclosing parasites. The red corpuscles in the phagocyte cells were observed as hyaline discs, and the arrangement of pigment in the parasites contained in these decolorised red blood-cells was similar to that observed in infected corpuscles found free in the blood-vessels. Alongside of well-preserved macrocytes many necrotic cells were found with swollen nucleus and little chromatin. Few of the multinuclear white cells contained pigment. The small mononuclear cells (lymphocytes) did not contain pigment, nor did the Malpighian follicles, such pigment as was observed in them being evidently in the endothelium of the capillaries. The blood of the splenic vein, examined fresh, contained a large quantity of phagocytes laden with pigment. In the liver not much was found except pigment-carrying macrocytes occupying the lumen of the intralobular capillaries, pigmented endothelium of the vessels, and pigmented cells of Kupfer. There were fewer pigment-carrying macrocytes in the hepatic than in the splenic vein. Fragments of hæmoglobin were sometimes found in the liver-cells, their nature being evident when the tissue was examined fresh. Bignami remarks that the elimination by the liver of the coloured substance of the red corpuscle which has been killed by the parasite explains the polycholia observed by Kelsch, and probably explains the jaundiced appearance of many of these patients. In the kidney the changes are generally few. There is pigmentation in the glomeruli, sometimes due to large white cells filling the lumen of the capillary, and sometimes the pigment is in the endothelium of the vessel. Few parasites are found in the kidney, but in some cases there is coagulative necrosis of the epithelium of the convoluted tubes. This change is observed in distinct areas, diseased epithelium being sometimes found in close juxtaposition to epithelium which has a normal appearance. There are not more parasites found in such a case than in cases which have no such lesion, from which he infers that the lesions are probably due to toxic action.

In these cases it was found that the adult and sporulating forms of the parasite accumulate mostly in the capillaries and particularly in the brain where the lumen of these vessels is very small. Crescent forms in all their stages were mostly found in the spleen.

The rapidity of the symptoms is associated with the short cycle of the parasite, and the interference with the cerebral circulation sufficiently explains the predominance of cerebral symptoms. In his "Studi Sull' Anatomia Patologica della Infezione Malarica Cronica" ('Bollettino della R. Accademia Medica di Roma,' 1893), Bignami described the changes that take place as a result of malarial infection in the liver and spleen, his inferences being formed by a comparison of the data afforded by fatal cases in which the patient had succumbed at various intervals during and after the febrile process. It was found that as the immediate result of the infection, there is a deposit on a large scale in the spleen pulp of white corpuscles containing pigment, parasites, and altered red corpuscles. This deposit leads to the destruction of a large number of the proper elements of the pulp, which goes side by side with a process of regeneration. The pigment-bearing white cells are taken up by the lymphatics and accumulate in the periphery of the follicles, whence they find their way into the lymphatics of the septa, leading to the formation of lymphatic cysts, thickening of the fibrous tissue, and the formation of splenic tumours.

In this paper, Bignami explains that the arrest of the circulation by the pigment-bearing phagocytes leads to atrophy or necrosis of the liver-cells and how the cells may become charged with yellowish ochre-coloured pigment produced by the premature death of a large number of the red corpuscles. Proliferation of surviving hepatic cells, stellate cells, and endothelia leads to acute tumour and augmentation of function, polycholia. Only a small number of pigmented elements leave the liver, the pigment eventually being transported into the perivascular lymphatic spaces, whence enclosed in the white corpuscles

it is carried by the lymphatics to the periphery of the lobule. In three or four months after an acute infection it is found in the perivascular lymphatics of the capsule of Glisson.

Repair never takes place until all the pigment and parasitic deposit is cleared off.

As the pigment disappears and is not found in the neighbouring lymphatic glands, its destruction and elimination must take place *in situ*. It probably disappears by a process of oxygenation inside the white corpuscles.

If we compare the appearances found in the case which I have described with the summary which I have given of the results of Bignami's investigations, we see at once that they harmonise in every essential particular, and we are able to understand much of what had taken place in the history of the patient before his death.

The presence of pigment in the endothelia of the blood-vessels of the brain is referable to a generation or generations of parasites which preceded the one which filled the capillaries at the time of the man's death. Pigment-bearing cells in the spleen are also referable to these antecedent generations. The histological changes in the spleen and liver described by Bignami as following the acute processes were not found, and there is nothing which I have described that is necessarily referable to any attack of malarial infection preceding the three days of acute illness which was followed by death. The accumulation of pigment-bearing cells in the pulp of the spleen, their tendency to accumulate round the Malpighian follicle, and their presence and mode of arrangement in the liver, coincides with what Bignami has described as being found in death after an acute attack. The limited extent to which the cells of the lobules of the liver have undergone a necrotic process bears out the same view, whilst in none of the sections which I examined were any appearances observed which indicated the beginning of the regeneration of hepatic tissue.

The appearances presented by the parasites in the

brain and the blocked condition of the cerebral vessels correspond in every detail with the appearances described by Bignami. A similar remark is applicable to the condition of the kidney. The necrosis of the epithelium of the convoluted tubes is similar to that described by Bignami as being found in a certain number of the cases of fatal pernicious fever in Italy.

But not only are the changes found in the organs similar to those described by the Italians, but the appearances presented by the parasite itself are identical with those described as characteristic of the parasite of Marchiafava. This will be best understood by comparing the drawings which accompany this paper with those published by Marchiafava and his pupils.

It seems, therefore, highly probable that at all events one form of the pernicious parasite of the West Coast of Africa is identical with the parasite of the summer-autumn fever of Italy. That the same variety of parasite may produce results of varying severity according to circumstances is a fact already well known. The parasite of tertian fever (*Hæmamœba vivax*), for example, produced more severe symptoms in Spain at one season than another (Dr. Marshall). As is stated in the paper by Dr. Marshall and myself, published in this volume, the attacks which it produces are mild in spring and early summer, increasing in severity as the hot weather comes on, until in August and September they may cause very severe and sometimes pernicious attacks. Similarly, it may be due to the local conditions prevalent on the West Coast of Africa that *Hæmamœba præcox* produces so frequently fever of such a severe type. There are cases of fever of milder types in the same district, which I have reason to believe, from private information which I have received, are due to forms of parasites similar to forms observed in Europe in the fevers which are usually of a comparatively mild type, but the facts from which I have gathered this impression have not yet been published by my correspondent,

and the information given me is not sufficiently full to warrant the expression of an opinion of a decisive character.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. viii, p. 132.)

formed needed no treatment. As it produced no symptoms and did not interfere in any way with her duties, for several years she ignored it, and sought no advice until three years ago, when she began to have occasional attacks of pain over the liver, followed by vomiting and slight yellowness of the skin, though she said that this never amounted to true jaundice. Her health began to fail, and she began to lose flesh four months ago, but the tumour did not appear to alter materially until six weeks before I saw her, when, whilst in the act of stooping to pick up a bottle, she was suddenly seized with an acute pain in the tumour, which straightway began to increase and to be painful and tender. From this time her illness had been continuous, and she had been confined to bed or the couch.

When she saw me, the right lobe of the liver reached to the level of the umbilicus, and from its lower border a firm rounded tumour passed down as far as the right groin. The tumour was dull on percussion, and did not fluctuate, though a thrill on succussion showed the contents to be fluid or semi-fluid. The left lobe of the liver was not enlarged materially, and no nodules could be felt. A diagnosis of enlarged gall-bladder due to impaction of gall-stones in the cystic duct was made, and the presence of secondary malignant disease was suspected.

Operation was advised, and the patient was admitted to a surgical home, the following operation being performed November 23rd. The abdomen was opened by an incision in the right linea semilunaris, when the gall-bladder was found to be adherent to the parietal peritoneum, as well as to the liver, stomach, omentum, colon, and small intestines.

Aspiration removed about two ounces of muddy-looking fluid, but the greater part of the contents of the gall-bladder was of putty-like consistence, and had to be removed by a lithotomy scoop after incision of the sac. One gall-stone, the size of a cherry, came from the interior of the gall-bladder, the walls of which were thickened

and infiltrated. The adherent omentum was ligatured off and other adhesions were separated, when it was found that the entrance to the cystic duct was almost occluded by a growth the size of a walnut, and beyond this could be felt a rounded gall-stone the size and shape of a thrush's egg. In consequence of this discovery, I decided to perform cholecystectomy, and proceeded to detach the gall-bladder from the liver, when I discovered a whitish nodule on the under surface of the right lobe of the liver, clearly an extension from the growth in the cystic duct.

It now became manifest that it would be useless to remove the one without the other; I therefore at once decided to attempt the removal of the affected portion of the liver, and to this end I had two knitting-needles boiled, and selected a piece of non-fenestrated drainage tube to act as a tourniquet.

I prolonged the incision quite up to the costal margin, had the right lobe of the liver and remains of the gall-bladder dragged forward through the wound, and encircled the protruding part with the rubber tube, which after encircling it twice was tied and the ends cut off. The tourniquet passed beneath the gall-stone, and therefore well beyond the growth in the cystic duct as well as the nodule in the liver. In order to prevent the improvised tourniquet from slipping, I pushed the knitting-needles through the liver and cystic duct transversely just above the elastic, leaving the ends resting on the skin on either side of the stump. There was then no difficulty in tucking down the parietal peritoneum and suturing it to the visceral peritoneum on the under surface of the liver and duct below the level of the tube. Sponge pressure, aided by a few catgut ligatures, soon arrested the bleeding from the torn adhesions, and after wiping out the abdomen the wound was closed layer by layer, leaving the disease external at the upper part of the incision.

The projecting lobe of liver, together with the remains of the gall-bladder and cystic duct, were then cut away half an inch above the needles, there being absolutely no

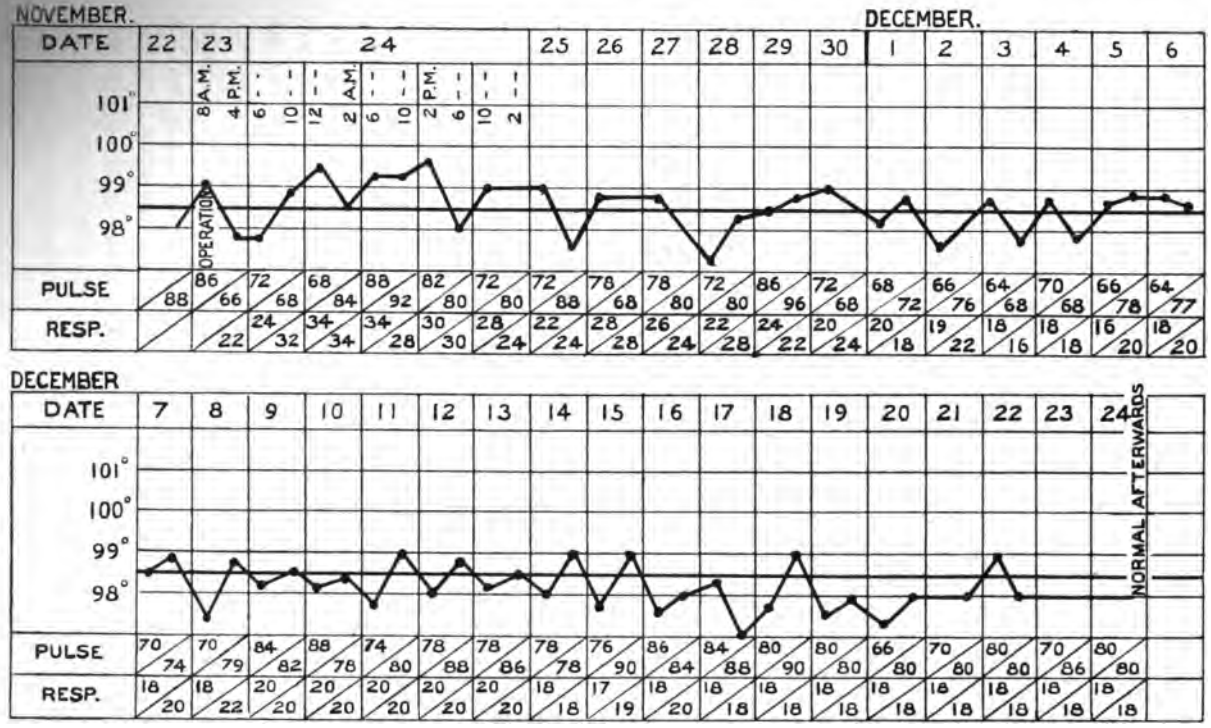
bleeding and apparently very little tension. The stump was dusted with boracic powder, and was dressed daily in order to keep it as dry as possible.

The wound beyond the stump healed by first intention, and the stitches were removed at the week end. The first needle came away at the end of a fortnight, the second a week later, and the stump beyond the ligature, together with the ligature itself, was removed within the month. After the separation of the slough, the granulations exuded a little bile for about ten days. The patient was on the sofa at the month end, and returned home six weeks after operation looking and expressing herself as quite well. She had lost her anxious look and had gained flesh. Her appetite was good, and her digestion seemed perfect.

The original and unaltered temperature chart appended shows that the recovery was absolutely uneventful: the pulse never exceeded 90; the temperature was never below 98° or above 99·2°, and the patient did not vomit even once,—in fact recovery was as smooth as after an ordinary ovariotomy. There was absolutely no shock.

The operation, in which I was assisted by Mr. Towers, occupied about an hour and a quarter, and the patient took the C. and E. mixture most comfortably, it being administered by Dr. McGregor Young.

Remarks.—Although hepatectomy for a pedunculated lobe, or for a well-defined and separable tumour, is known to be within the range of modern surgery, until the present case I scarcely thought it probable that hepatectomy for cancer would come within the scope of practical operations. When, however, I found that the remainder of the liver was free from disease, I felt that I ought to give the patient the chance of cure by attempting the removal of the disease, although the precedent was wanting; and in this Dr. O'Connell, who was present, fully agreed. With regard to the method employed, it was so simple and proved so effectual, that although thought out on the spur of the moment additional consideration has



DATE	22	23	24						25	26	27	28	29	30	1	2	3	4	5	6
		8 A.M.	6 -	10 -	12 -	2 A.M.	6 -	10 -	2 P.M.	6 -	10 -	2 -								
		4 P.M.																		
PULSE	88	86 / 66	72 / 68	68 / 84	88 / 92	82 / 80	72 / 80	72 / 88	72 / 68	78 / 80	78 / 80	72 / 80	86 / 96	72 / 68	68 / 72	66 / 76	64 / 68	70 / 68	66 / 78	64 / 77
RESP.		22	24 / 32	34 / 34	34 / 28	30 / 30	28 / 24	22 / 24	28 / 28	26 / 24	22 / 28	24 / 22	20 / 24	20 / 18	19 / 22	18 / 16	18 / 18	16 / 20	18 / 20	

DATE	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
PULSE	70 / 74	70 / 79	84 / 82	88 / 78	74 / 80	78 / 88	78 / 86	78 / 78	76 / 90	86 / 84	84 / 88	80 / 90	80 / 80	66 / 80	70 / 80	80 / 80	70 / 86	80 / 80
RESP.	18 / 20	18 / 22	20 / 20	20 / 20	20 / 20	20 / 20	20 / 20	18 / 18	17 / 19	18 / 20	18 / 18	18 / 18	18 / 18	18 / 18	18 / 18	18 / 18	18 / 18	18 / 18

not led me to think it necessary to advise a modification of the procedure for any similar case in the future.

The tumour after removal weighed half a pound, after the moisture had drained away, and this did not include either the pultaceous material removed from the gall-bladder or the walls of the gall-bladder, except that portion attached to the liver itself. Mr. J. H. Haigh reports the growth to be epithelioma. For the microscopic characters of the new growth *vide* Plate III.

This case, along with many others that have come under my observation, amply demonstrates the causative relationship of cancer to gall-stones, and I think it teaches us the lesson that simple enlargement of the gall-bladder, even unaccompanied by symptoms, ought not to be left without operation, which, with proper precautions and in skilled hands, is almost devoid of danger.

ADDENDUM, MARCH 10TH, 1896.

I had the opportunity of seeing the patient ten days ago, and of carefully examining the region of operation; so far as I could say there was no sign of recurrence of disease, the patient being of a good colour, and the remains of the liver, so far as could be ascertained by palpation and percussion, remaining normal in size and being free from tenderness.

I was asked to see her on account of a small swelling, situated in the abdominal wall, just above the inner end of the right Poupart's ligament, which was thought to be increasing in size, and which was causing uneasiness from the fear that it might be a secondary growth, though situated so far from the original site of the disease. I thought it better to remove it, and on incision I found the tumour to consist of an encapsuled swelling in the abdominal wall, between the skin and the aponeurosis of the external oblique, resembling a sebaceous cyst, in that the cyst wall was quite distinct and the contents were

of putty-like consistence. Under the circumstances I removed the tissues immediately surrounding the capsule as well as the tumour itself. The wound healed by first intention, and the patient was up on the third day.

Mr. Stott, pathological curator at the infirmary, informs me, after having examined the parts removed, that he can find no sign of morbid growth, and that in structure it resembles a sebaceous cyst.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. viii, p. 156.)

and extension of right elbow feeble. Movements of right shoulder fairly good. Movements of right leg weaker than those of opposite side. Boy too drowsy to be carefully examined as to sensation. No optic neuritis. Complains of intense pain in the back of the head and neck, also of frontal and vertex headache. Dressings as before.

31st.—Patient nearly comatose and apparently dying.

Operation (9 p.m.).—Dr. Buxton gave chloroform. The scalp having been carefully shaved and cleansed, a large ∩-shaped flap about five to six inches in diameter was thrown down so as to include in the bone exposed both trephine holes. The scalp was carefully peeled off from the masses protruding through the openings in the skull. It was soon apparent that the mass coming through the posterior or original trephine opening was a soft tumour, not brain tissue. A dura mater elevator having been passed from one trephine opening to the other, the bone between was removed, and an opening in the skull was obtained about 3½ inches by 2 inches. The dura was now incised, and what appeared at first sight to be normal compressed cortex was exposed. The anterior hernia was now ligatured with silk and removed carefully. The posterior hernia was evidently a part of a tumour, and on beginning to separate it posteriorly from the cortex it was found that all the part exposed by the removal of bone between the trephine openings (which was previously taken to be compressed cortex, and which it exactly resembled in colour, and in apparently having convolutions and a pia with vessels—see Fig. 2) consisted of tumour substance, and that it could easily be separated from the brain beneath by the finger; this was accordingly done. The tumour removed is shown in Figs. 3 and 4. One piece of the tumour extended down on to the orbital plate of the frontal bone lying between the dura and what was apparently the third frontal convolution. This piece was very diffluent and difficult to manage with the fingers, so Dr. Head kindly boiled a silver egg-spoon, and with this instrument its removal was easily effected. On

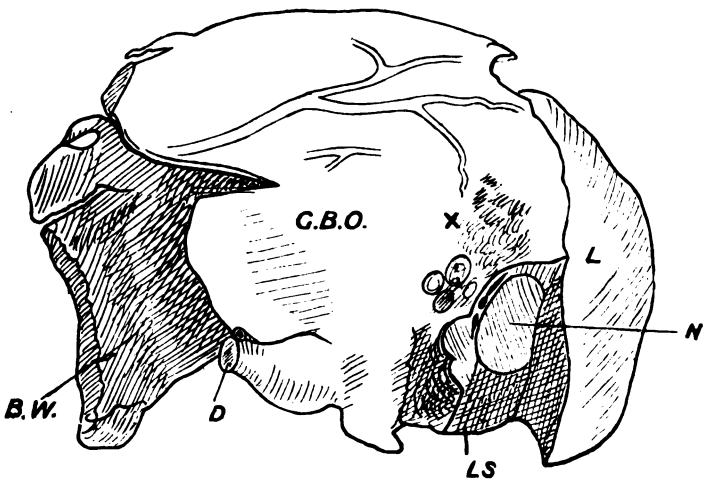


FIG. 1.

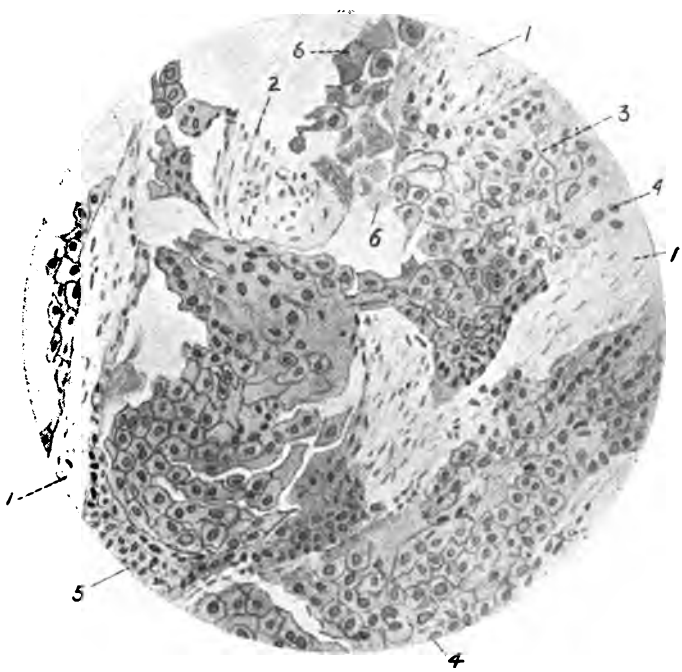


FIG. 2.

Imp. Adlard and Son.

DESCRIPTION OF PLATE III.

Excision of a Portion of Liver for Tumour (MAYO ROBSON).

FIG. 1.—(Reduced $\frac{1}{3}$.) G.B.O. Outer surface of gall-bladder; near × the growth is infiltrating the wall, shown in shaded portion. B.W. Thickened and infiltrated wall of gall-bladder, laid open. L. Liver. L.s. Liver laid open to show—N, Secondary malignant nodule in liver; D, Cystic duct.

FIG. 2.—Microscopic section of the new growth. (Hartnack Obj. No. 4, oc. 3 = × 120. Drawn by J. W. Haig.) 1, 1, 1. Connective tissue of alveolar walls. 2. Connective-tissue nuclei. 3. Epithelial cells, somewhat squamous in appearance. 4, 4. Nuclei of ditto. 5. Smaller rapidly growing cells of basement layer. 6, 6. Degenerated epithelial cells in which the nuclei have disappeared.

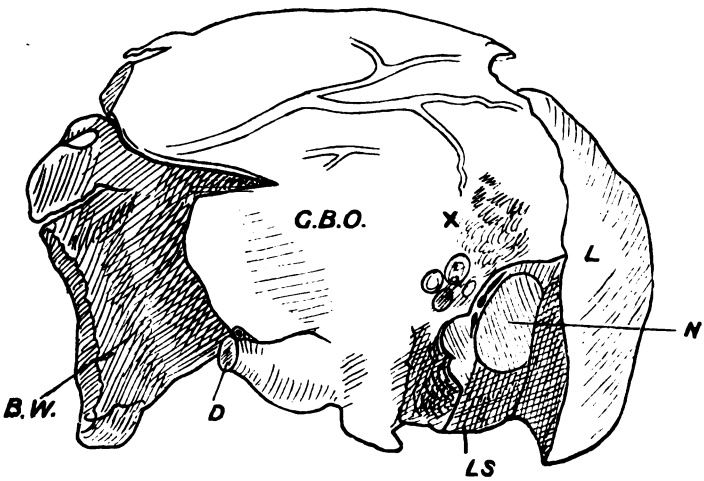


FIG. 1.

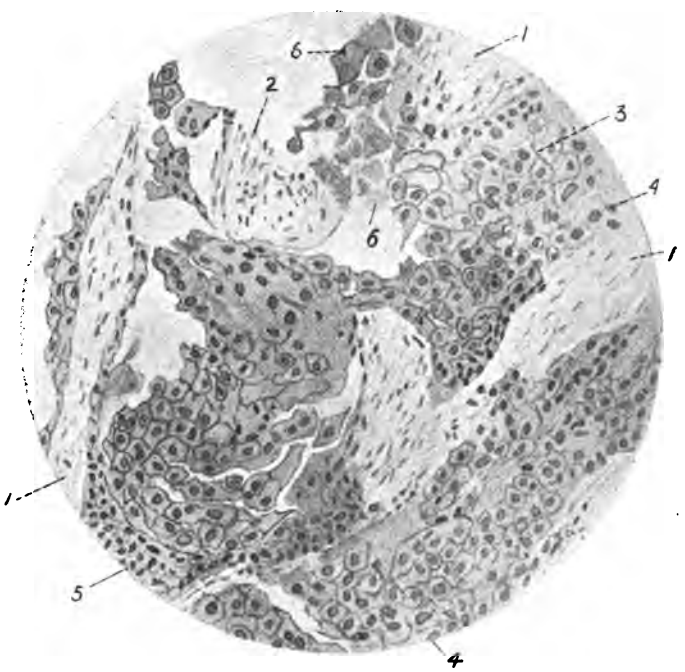


FIG. 2.

Imp. Adlard and Son.

unconscious, and had a general epileptic attack. The friends principally noticed the convulsion in the left arm; the eyes were turned strongly upwards; the tongue was not bitten, neither did he pass water. The fits are said to have lasted twenty minutes. In June, 1890, when the fits ceased, some weakness of the right side of the face was noted, and, moreover, speech became affected; the lower jaw was noticed to drop as if weak, and the mouth was not closed properly. Six weeks ago the right arm and leg became weak; this weakness has gradually become more marked, and coincidently with this speech has improved, and the dropping of the lower jaw is less. For between two and three months there has been frontal headache and occasional vomiting, the latter symptom being more marked during the last few weeks.

Previous history.—Measles when quite young; no other illness. Has always been thin and pale.

Family history.—Maternal grandfather died of phthisis; no other fact of importance elicited.

Present condition.—A pale, weakly-looking boy, very thin. Head: no tender spot and no scar on scalp discovered. Head and trunk movements normal. Face: lower part of right side does not move as well as left. Slight difference in action of corrugators. Eyes shut equally. Weakness of right side marked in smiling or in showing teeth. Tongue protruded straight. Palate: right side does not move as well as left. Eyes: movements good; no nystagmus; pupils equal, and act normally to light and accommodation. Ophthalmic examination: right, some blurring of margins of disc, especially at inner side; left disc swollen, edge invisible. There is considerable myopia. Speech: no appreciable defect. Arms: right colder than left, and of a bluish colour. Dynamometer: R. = 0, L. = 25. Much weakness of movements of right arm at shoulder, and complete loss of power of movement at elbow, wrist, fingers, and thumb-joints. No rigidity: muscles small and flabby. No

localised atrophy. *Left arm*, no loss of power. Wrist-jerk active, no marked difference on the two sides.

Lower extremities.—Movements of right hip- and knee-joints feeble. Movements of right ankle and toes absent. Right limb colder than left. Muscles flabby ; no rigidity. Knee-jerks active ; right more brisk than left, and with slight patellar clonus. No ankle-clonus. Plantar, cremasteric, and abdominal reflexes not elicited. Left lower limb normal. Gait : drags right leg much in walking. Sensation : no affection or disturbance of sensation. Micturition not affected ; urine normal. Defæcation normal. Heart, lungs, and abdominal organs normal.

Electrical examination.—Muscles react normally to faradic and galvanic currents.

Aural examination.—R. M.T. opaque, retracted somewhat ; cone of light interrupted. L. M.T. not so opaque, retracted anteriorly and below handle of malleus. (These signs point to old catarrhal mischief in both ears.) Bone conduction normal.

Progress. — October 13th, 1890.—Complains occasionally of attacks of sharp pain across frontal region. Vomited yesterday, the first time since admission. The vomiting was purposeless, but did not occur during an attack of pain.

28th.—Since last note many attacks of vomiting have taken place. Optic neuritis in both eyes more advanced.

November 7th.—Severe headache and vomiting. Optic neuritis more intense. The head was shaved and cleansed to-day ; the position of the median line and left sulcus of Rolando marked on the scalp, and an antiseptic dressing applied.

8th. *Operation.*—Chloroform was administered. A Horsley's ∩-shaped flap (see Fig. 1) was cut and thrown downwards with the periosteum. After the bleeding was arrested the flap was wrapped in warm and wet antiseptic gauze. The trephine was then applied to the parietal bone over the lower part of the motor cortex, and a disc of bone one inch in diameter removed. The pin of the tre-

leg (slight right hemiplegia). This does not interfere with his getting about very well, and with an apparent attainment of almost complete recovery. The flap is concave, and the hair has grown long over it.

Readmitted September 18th, 1893. The boy had been quite well up to about three weeks ago, when the father noticed that the scalp over the aperture in the skull, which had up till then remained concave, began to be convex and to bulge.

There were no other symptoms, no pain, no vomiting. He would, however, frequently cry out in his sleep and talk in his sleep a great deal, which he previously had not been accustomed to do.

One evening ten days ago he became very drowsy and emotional, and said he was going to have a fit. He did not have a fit, nor has he had any up to the time of his admission.

The scalp flap, however, gradually bulged more and more; the slight weakness fo the right side, however, did not appear to increase.

Condition on admission.—General condition fair. Complexion decidedly anæmic. *Nervous system.*—Mental condition dull. No delusions or hallucinations. Sleeps fairly well. No headache. *Motion: arm.*—All movements of left arm and leg good. Movements of right shoulder and elbow good, but weaker than on left side: complete wrist-drop. Flexion of fingers fairly good. No power of extension. Little or no rigidity of arm. No wasting. *Leg.*—Movements of the right hip and knee are fairly good. Movements of the right ankle and right toes are practically lost. No wasting or rigidity. *Sensation.*—There is marked impairment of the sense of touch over the whole of the right side of the body. This is most marked on the right arm. Localisation of tactile sensations on right side is most inaccurate. Painful sensations on the right side are not appreciated as they are on the left. *Reflexes.*—Right knee-jerk +. Ankle-clonus right side. Right plantar brisker than left.

Abdominal present. *Cranial nerves.*—Marked paralysis of lower half of right side of face. No affection of any other nerve. Tongue comes out straight. Speech slow.

Ophthalmoscopic examination.—Discs and fundi in same condition as last note. No optic neuritis.

There is great bulging of the scalp over the opening in the skull (see Fig. 6). The bulging mass fluctuates and pulsates. The scars of the old operation wounds are healthy, but look red and more vascular than usual. In the scalp, over and around the tumour, numerous veins are visible.

FIG. 6.

From a photograph. Appearance of patient in September, 1893. Scalp flap bulging.

It was thought that a tumour had again grown between the dura and cortex, and it was determined that an attempt should be made to remove it.

September 28th.—*Operation.*—Chloroform was administered at 9 a.m. The old scar was incised and the flap turned down in the usual way. There was free hæmorrhage from the scalp, and the bone bled very freely.

The bone was noticed to be of a pink colour, and on it minute pink lines indicating the course of vessels could on close inspection be observed. The bulging mass was found to be part of a large tumour, and it was then determined to enlarge the opening in the skull, and try if possible to remove it. On commencing to remove the bone with cutting forceps the bleeding which ensued was persistent and uncontrollable; the bone surface literally wept blood, and the operation was promptly abandoned. Nevertheless the hæmorrhage had evidently had a considerable effect on the patient, and he suffered much from shock. The scalp flap was stitched rapidly with horsehair in place, the dressing applied, and warmth and restoratives used. The temperature was 95° F., and the pulse 140. The boy, however, did not rally; he was exceedingly restless, and towards evening the respiration became sighing, and the pulse at the wrist could only just be felt. Temperature 96° F., pulse 144. At 9 p.m., the condition remaining the same, it was determined to infuse him with warm normal saline solution. This was done, but without effect, and death took place at 10 p.m.

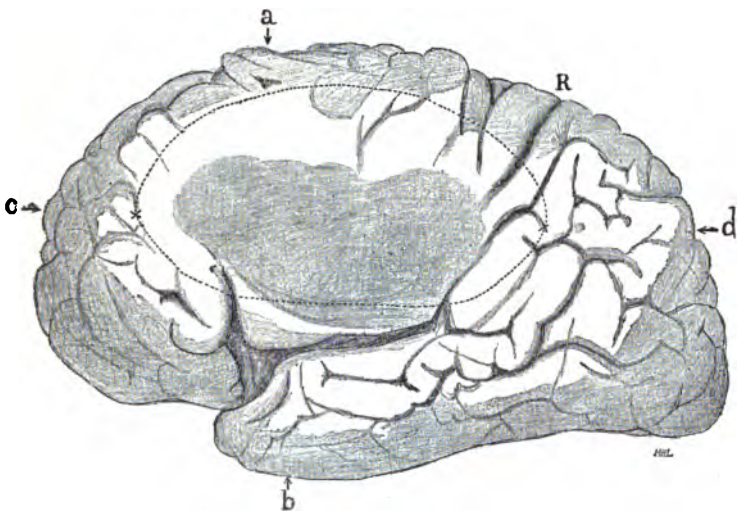
(N.B.—During nearly the whole period of the illness the temperature of the right or paralysed side was slightly higher than the temperature of the left. For some three or four months after the first operation the reverse was [speaking generally] the case. After the first operation, when pressure was relieved the pulse was not slow. It was seldom more or less than eighty beats per minute.)

Post-mortem examination by Dr. Colman (September 29th, 1893).—Body fairly nourished. Recent operation wound on left side of head. The tumour which had been so prominent during life (see Fig. 7) had now sunk, and was level with the surface. The skull-cap has a large aperture on the left side, which has been recently enlarged; it came away easily from the dura mater.

The dura mater was not adherent to the convolutions

except close to the site of the tumour. The tumour was partly superficial and partly subcortical, as shown in Figs. 8 and 9. Where it was subcortical the convolutions were broadened out and pale. Where it involved the surface the outline was uneven, and its substance was very soft to the touch.

FIG. 7.

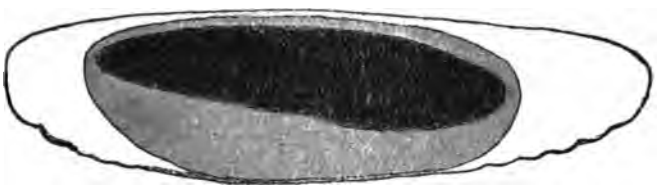


View of brain at autopsy. (Half natural size.)
Dotted line shows roughly extent of subcortical tumour. Within the dotted line the white surface shows the flattened convolutions; the grey shading represents adherent dura mater.
R = sulcus of Rolando.
a, b, shows line of section represented in Fig. 9.
c, d, shows line of section represented in Fig. 8.

On separating the hemispheres a cyst or cavity in the tumour was found bulging into the lateral ventricles (see Fig. 9), its wall being formed merely by thickened ependyma. There was no communication with the ventricle. On cutting into the cyst a thin brownish fluid escaped. The "cyst" was found to correspond pretty well with the tumour on the surface of the brain, and was bounded by a soft sarcomatous mass, which was thickest at the posterior part of the cyst The tumour and "cyst"

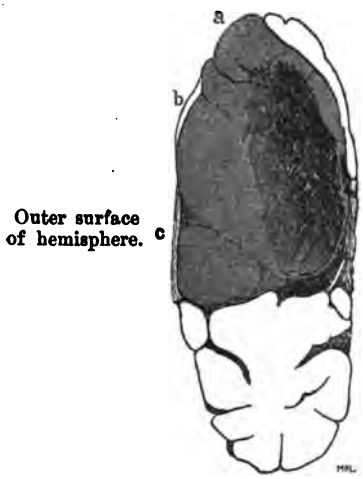
appeared to involve all the white matter of the centrum ovale as far down as the basal ganglia.

FIG. 8.



Longitudinal section at *c*, *d*, in Fig. 7. (Half natural size.)
Shaded area = tumour.
Black centre = cavity in tumour.

FIG. 9.



Transverse vertical section at *a*, *b* in Fig. 7. (Half natural size.)
Shaded area = tumour.
Black centre = cavity in tumour which does not communicate with lateral ventricle.
a = cortex destroyed by tumour.
b = cortex compressed by tumour.
c = adherent dura mater.
This drawing shows the destruction of the corona radiata by the tumour.

The rest of the brain was quite natural.

Spinal cord.—No naked-eye change in fresh condition.

Microscopical examination.—Sections showed degeneration of the crossed pyramidal tract on the right side, and to a slight extent on the left. The direct pyramidal tracts were not affected at all.

No new growth was found in any of the body organs, which were quite healthy.

The tumour was an angiosarcoma. In places it was almost entirely cellular, in others almost completely vascular, and in other places there were vessels of considerable size, with thin walls separated by sarcomatous tissue.

REMARKS.

		PAGE
1.	Abstract of Notes	189
2.	Diagnosis	191
3.	Brain Tumour and Trauma	191
4.	Intermission of Symptoms	192
5.	Twitching of the Index Finger	192
6.	Moral Degradation	193
7.	Surface of Tumour mistaken for Surface of Cortex	193
8.	Enormous Extent of Tumour; Symptoms slight	193
9.	Cranio-cerebral Topography	194
10.	Platinised Silver Tubes for Drainage	194
11.	Hæmorrhage from Bone	195
12	Was the proper Surgical Treatment adopted ?	195
13.	Surgery and Tumour of the Brain	196

1. Abstract of the Notes.

The story of the case may be briefly summed up as follows:—In December, 1889, the boy was stunned by a blow on the head, and between this date and his admission into the hospital, in October, 1890, he suffered successively from the following symptoms:

1. Irritable temper.
2. Fits commencing with twitching of the right angle

of the mouth, and followed by paralysis of the lower part of the right side of the face.

3. The gradual extension of the paralysis to the right upper and lower extremities.

4. Slow speech.

5. Severe frontal headache.

6. Purposeless vomiting.

7. On admission double optic neuritis was observed.

The symptoms increasing in severity after a month's residence in the hospital, the lower part of the left motor cortex was exposed by operation (November, 1890), and a subcortical "cyst" tapped, the result being that the symptoms practically disappeared.

Two months later (February, 1891) the symptoms are noted to have gradually returned, hence a tube was passed into the "cyst" in order to effect permanent drainage. In June, 1891, the tube was removed, and the following month the boy left the hospital apparently quite well, except for a very slight right hemiplegia.

In May, 1892, he was readmitted with marked right hemiplegia, and in July a drainage-tube was again passed into the "cyst," but without causing the same complete relief from symptoms as on the previous occasion.

The boy remained in hospital during the succeeding six months, his condition varying much, and being better or worse according as the intra-cranial "cyst" drained freely or not through the drainage-tube.

In January, 1893, the state of the patient became more critical, and at an operation which was then performed a tumour three ounces in weight was removed from between the dura and the motor cortex of the left cerebral hemisphere.

In June, 1893, the child left the hospital with the scalp flap healed and concave, and in fair general health. There was still slight right hemiplegia.

In September, 1893, he was again readmitted with a recurrence of the symptoms, and with the scalp flap bulg-

ing. An operation was attempted, but death occurred from uncontrollable bleeding from the bone.

There are many points of interest in the diagnosis, symptoms, pathology, and treatment of this case, to which reference may be made.

2. Diagnosis.

The history of the case clearly pointed to a diagnosis of brain tumour; and the symptoms showed that the tumour occupied a definite position, and involved the cortex or corona radiata immediately beneath the cortex.

It was hoped at first, notwithstanding the extreme rarity of subcortical traumatic cysts, that the "cyst" might be hæmorrhagic in origin, but the gradual progress of the case in the direction away from recovery, and, indeed, the absence of cyst-wall and the nature of the fluid (really plasma) removed, negatived the hopeful view, and made for the diagnosis of malignant growth. The notes say when the boy was first admitted to the hospital that "there is no affection or derangement of sensation," but this statement is not so satisfactory as a positive assertion that "there is no defect in the localisation of tactile sensations, and no loss of muscular sense on the paralysed side." I do not remember whether this was so or not, but seeing that the cortex was involved (by pressure at least) it is not improbable that these signs were present. The absence of marked sensory disturbance on the paralysed side would suggest that a tumour occupying the corona radiata was nearer the cortex than the internal capsule.

3. Brain Tumour and Trauma.

A history of injury to the head may sometimes be obtained in cases of tumour of the brain. In one case of glioma of the occipital lobe on which I operated, a healed fracture of the skull three inches long was exposed on

turning down the scalp flap, and this fracture was over the site of the tumour. There was no scar on the scalp, and no history of injury had been given by the patient or his friends.[1]

4. Intermission of Symptoms.

This occurrence is by no means uncommon in the early stages of brain tumour. In this case in June, 1890, there was weakness of the right side of the face with jaw-drop and slow speech. In July, 1890, weakness of the right arm and leg became a marked sign, and coincidently there was a return to normal in the power of speech, and recovery from jaw-drop.

5. Twitching of the Index Finger.

A movement of the index finger, alone or in conjunction with a movement of the thumb, can be produced by stimulation of the cortex both in *Macacus rhesus* and *Macacus sinicus*. During the autumn of 1890 the boy had movement of the index finger at the commencement of a fit, so that it is clearly true to affirm that there is an area on the human motor cortex (as in monkeys)[2], where the movement of the index is more highly represented than any other movement. "Jaw-drop" (depression of the lower jaw) can also easily be elicited by cortical stimulation in monkeys.

[1] In Dr. Hadden's case (referred to later on in a note) there was a history of trauma; in Dudley's case ('Brain,' vol. xi) a cerebral tumour was apparently the direct result of a cranial injury.

[2] "Experimental Observations of the Brain of the Monkey," Hadden and Ballance, 'St. Thomas's Hospital Reports,' vol. xix. See also the classical papers by Beevor and Horsley, "A Further Minute Analysis of the Motor Region of the Cortex Cerebri of the Monkey," 'Phil. Trans.,' 1888, vol. clxxix, p. 232, "Centre," 85; "Analysis of the Motor Cortex of the Orang-outang," 'Phil. Trans.,' 1890, vol. clxxxi (B), p. 140. These experimenters locate the "centre" for the movements of the index immediately above the area on the ascending parietal gyrus where the movements of the thumb are most highly represented.

6. Moral Degradation.

Previous to the removal of the large intra-dural tumour the patient became addicted to stealing in a most artful and cunning manner. Dr. Jackson,[1] in the discussion on "imperative ideas," said, "The degree of mental degradation will, I think, be helpful in gauging the patient's condition, especially with regard to the degree of raised intra-cranial pressure from the tumour." It may be stated that in this case, with an opening in the skull and dura, the general intra-cranial pressure could not have been much raised. Would it have been right, then, to diagnose considerable local pressure (which was actually present) from the signs of moral degradation?

7. Surface of Tumour mistaken for Surface of Cortex.

The tumour removed at the operation in January, 1893, when first it came into view, was mistaken by all those present for bulging cortex. The cystic character of the growth gave the false appearance of sulci and convolutions, and its covering of a delicate membrane with vessels, indistinguishable from pia mater, aided in making the distinction between tumour surface and cortical surface impossible until a large opening had been made in the skull. Possibly some of this vascular enclosing membrane was pia, which had been pushed away from the cortex as the fungating tumour grew out through the hole made by the drainage-tube. At the autopsy, nine months after this operation, no sign of tumour growth was found between the dura and the cortex, so that the tumour would seem to have been completely removed.

8. Enormous Extent of Tumour;[2] Symptoms Slight.

The tumour probably commenced in the frontal lobe and grew very gradually backwards, pushing before it the

[1] 'Brain,' part lxx, p. 321.

[2] Sometimes very large tumours only produce symptoms shortly before

pyramidal fibres of the corona radiata (see position of sulcus of Rolando in Fig. 7). The slow growth of the tumour, the large opening in the skull, and the possible vicarious employment of the other hemisphere are points which offer a possible explanation. The idea that drainage of such a malignant cyst will retard or arrest its growth is probably founded on an erroneous conception of the pathology of malignant tumours.

9. Cranio-cerebral Topography.

Only a disc of bone was removed at the first operation, and the place to apply the trephine was previously determined on by marking the scalp by means of one of the many methods of cranio-cerebral topography. In certain cases the methods which depend on scalp markings are no doubt useful. It is to be remembered that these markings are lost as soon as the scalp flap is made. The operator should rely, then, not on scalp markings, but on the known relations of the sutures of the skull to the fissures and convolutions of the brain.

10. Platinised Silver Tubes.

The use of platinised silver tubes for drainage has certain distinct advantages. I learnt their use from my friend Mr. R. H. Clarke, who employed them in the treatment of roaring in horses. Whereas silver tubes become thin by erosion and discoloured, tubes coated with platinum keep their polish and colour, and are indeed quite unaffected by contact with living tissues and

death, *e.g.* the case referred to on page 24 of Gowers' 'Clinical Lectures' became suddenly ill six weeks before death; the brain is in the museum of the National Hospital. A still more remarkable case was published by my friend and colleague, the late Dr. Hadden, in 'Brain,' vol. xi, p. 523. The fatal illness of the patient commenced thirty-six hours before death. He had suffered previously from occasional epileptic seizures. At the autopsy an enormous tumour was found in the right frontal region of the brain.

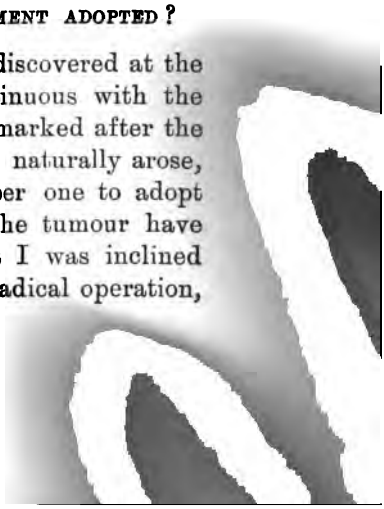
fluids. Conversely, platinised tubes cause no irritation of the tissues in contact with them. The difference between platinum and silver in this respect can only be appreciated by those who have observed the behaviour of both metals.

11. Hæmorrhage from Bone.

The patient died in consequence of hæmorrhage from bone at the last operation. It is quite possible that he would have lived some time longer if I had been content merely to pass a trocar through the scalp and withdraw the fluid from the cavity in the tumour. It is important to avoid the tendency of attempting too much. The local condition had much altered since the boy had been in the hospital some months previously. The scalp over the opening in the skull was pierced by new and large veins, and in exposing the bone it was discovered to be of a distinct pink colour for at least one inch all round from the margin of the opening. This vascularity cannot be explained by an appeal to the changes which occur in the healing process, and I believe it was due to an infection of the cut edges of the bone by the intra-dural malignant tumour removed at the operation nine months before. The tumour itself showed on microscopical examination in some places nothing but a network of vessels. Antiseptic wax was useless, and the Liq. Ferri Perchlor. fort. was scarcely more effectual.

12. Was the Proper Surgical Treatment adopted?

Perhaps the most remarkable fact discovered at the autopsy was that the tumour was discontinuous with the brain substance, and this was still more marked after the brain had been hardened. The question naturally arose, was the treatment by drainage the proper one to adopt when the case was first seen, or could the tumour have been shelled out and removed? At first I was inclined to blame myself for not attempting the radical operation,



but there are several considerations which now seem to support the view that the right treatment was adopted.

1. At the operation the interior of the "cyst" did not show any indication of a cyst-wall; indeed, the interior looked exactly like healthy brain substance, and the cortical surface when collapsed looked normal, and showed no line of junction between healthy and diseased tissue.

2. It is probable that what appears so plain and easy to carry out on the dead and hardened brain may elude surgical observation on the living brain.

3. This year I have operated on a very similar case, and bearing in mind my previous experience I made a strenuous endeavour to discover the presence of tumour tissue surrounding the "cyst," but utterly failed to do so.

4. Any operation undertaken for the removal of the tumour might very possibly have caused permanent aphasia,—a disaster which, in my opinion, should negative in such cases any operation designed to do more than relieve symptoms.

13. Surgery and Tumour of the Brain.

The last question is, What did surgery do for the patient? The reply is satisfactory. Life was prolonged for the most part in a happy condition for three years. The sure advent of blindness from the optic neuritis was prevented; and the symptoms—headache, vomiting, fits, palsy, aphasia, and mental and moral degradation—were either completely obviated or markedly mitigated.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. viii, p. 160.)

ON THE EFFECTS PRODUCED

BY THE

RETENTION OF FOREIGN BODIES FOR LENGTHENED PERIODS

IN THE

BRONCHIAL TUBES

BY

RICKMAN JOHN GODLEE, M.S.
SURGEON TO UNIVERSITY COLLEGE HOSPITAL, AND TO THE HOSPITAL FOR CONSUMPTION, BROMPTON, &c.

Received January 3rd—Read March 24th, 1896

I HAVE selected the following cases as illustrating most of the characteristic features of this extremely troublesome affection; and, although I cannot pretend that the paper contains much that is new, I trust that the interest of the cases as well as their comparative rarity will make it not unworthy of the attention of the Fellows, especially as two of the patients have been under observation for a long time after the expulsion of the foreign body.

CASE 1.—H. C—, a boy now 16 years of age, was admitted under the care of Dr. Ringer into University College Hospital on January 5th, 1893, supposed to be suffering from chronic empyema. There was a large amount of extremely offensive expectoration, and pus was

obtained with an exploring needle. The physical signs, however, pointed to the existence of bronchiectasis on the left side, while the right side was but little if at all affected. I ventured to suggest the possibility of the trouble having arisen from the inspiration of a foreign body, and on questioning the boy—but not before—he told us that seven and a half years previously he had "swallowed" the peg of a peg-top. We discovered afterwards that obvious symptoms made their appearance eighteen months later, and that two years after the accident the expectoration became offensive.

Under the circumstances I thought it wisest, after removing a piece of the ninth rib opposite the place at which pus was obtained, to stitch the lung to the pleura, and leave the incision of the lung to a subsequent occasion. On the first attempt under chloroform I was not successful, but a few days later, when the boy was sitting up, and with his cavity full, I opened, without difficulty, a bronchiectatic cavity, but found no foreign body. The expectoration was reduced but not stopped by this procedure, and the boy was made more comfortable. Subsequently the following methods were attempted for reaching the foreign body:

1. Probing through the wound.

2. Tracheotomy. Searching the bronchus with probangs and wires. Inversion of the patient on several occasions.

3. A second incision was made nearer the spine, by which the healthy pleura was opened, and the lung explored with the finger both behind and on the under surface.

All these attempts were fruitless. I need not detail the symptoms which he manifested during his stay in the hospital; they were of the usual description—rises and falls of temperature, increase and diminution of expectoration, occasional and sometimes severe hæmoptysis.

Ultimately the boy was sent home, and many were sceptical about the correctness of the diagnosis.

In October, 1894, however, during a fit of coughing, the rusty peg of the peg-top came out through the wound, in which he still retained a drainage-tube, and which indeed he found a great comfort, and called his safety-valve.

For some time we heard discouraging accounts of the boy's progress. The removal of the cause of offence did not appear to make much difference to his symptoms. It is, therefore, gratifying as well as surprising to hear from Mr. T. Hopkin Davies, of Port Talbot, under whose care the patient now is, that his general health is excellent. The tube has been removed, there is no expectoration, and there has not been any hæmoptysis recently. The clubbing is less, and there is no enlargement of the liver and no albuminuria. The breathing is said to be clear over the front of the left side of the chest, but a few moist râles are to be heard in the left scapular region. The right side of the chest appears to be quite healthy.

From the point of view of self-congratulation the present is the time for publishing this case. More than a year has passed since the expulsion of the foreign body,—a very reasonable time, it may be said, in which to form an opinion as to the prospects of a cure. Attention to the two following cases, however, will show that it would be very rash to draw any general conclusion from this apparently very favorable result; rash, indeed, to conclude that even he is completely out of the wood.

Case 2.—E. S. W— (patient of Dr. Anderson, of Mirfield), in February, 1891, being then six and a half years old, inspired a small ivory knob into his left bronchus. It remained there till March 31st, 1891 (*i. e.* for eight weeks), and set up bronchiectasis in the left lung, with typical symptoms; but no indication was given by the physical signs of the situation of any one large cavity when I first saw him in March, 1894. The left side was then retracted, and there was deficiency

of resonance behind and at the side. The physical signs were most abnormal just below the angle of the left scapula. I did not advise surgical interference.

Since his return home intra-tracheal inhalations have been tried, but not very energetically. The expectoration is very fœtid, and amounts to about four ounces a day. He empties himself systematically three times a day. Clubbing of the fingers is well marked, but there is no albuminuria. There is still marked dulness on the contracted left side, most marked below the angle of the scapula, and shading off gradually. There are copious moist râles, and whispering pectoriloquy over the dull area. The right lung remains apparently unaffected.

Case 3.—In June, 1888, I saw, with Dr. Symes Thompson, Dr. Powell, and Dr. F. Hawkins, the son of Dr. Adams of Greenwich, who was then four years of age. He had a fœtid expectoration, the source of which was doubtful, from which he had suffered for about seven months. He had been seen by other physicians, and it was supposed that his trouble had originated with whooping-cough, and there was said to have been at one time consolidation of the base of the right lung. At the time there was deficient movement of the right side, and dulness from the seventh rib to the eleventh behind, extending laterally from the posterior axillary line to one inch from the spine. Vocal fremitus over this area was absent. Breath-sounds were sometimes almost absent, sometimes loud and of a tubular character, and there were numerous coarse râles. A few râles were heard upon the left side.

I thought it was a case of bronchiectasis, and asked about the possibility of the presence of a foreign body in a bronchus, but was unable to hear of any likelihood of such an occurrence having happened.

A puncture with an exploring trocar one inch below the upper limit of the dulness revealed nothing. Another, slightly higher, drew off a drop or two of offensive pus. It was so superficial, however, that I altered my diagnosis,

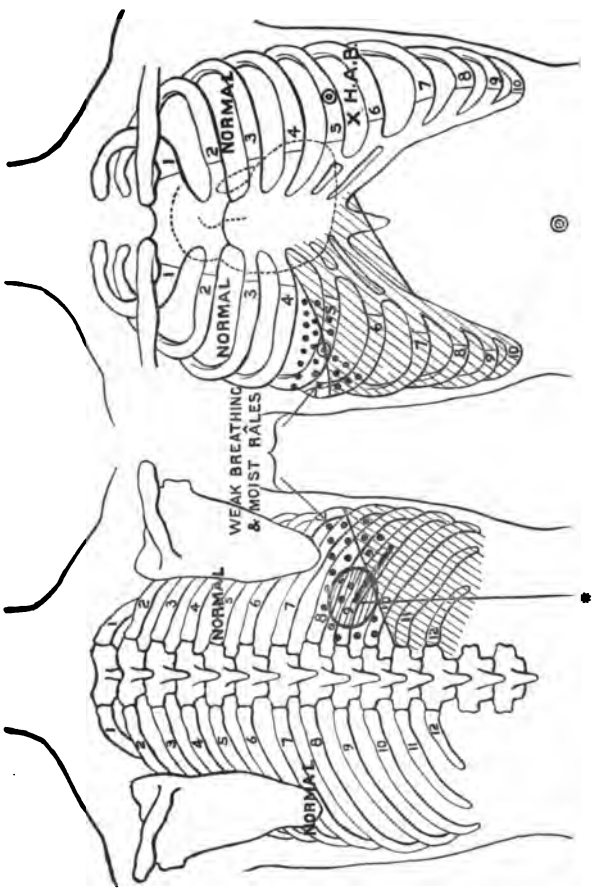
and after removing a piece of the ninth or tenth rib I incised the pleura before stitching up the lung to the chest walls, and found it, to my chagrin, free from adhesions. This necessitated a laborious stitching up of the lung, and probably the losing of the exact place at which the pus had been found. However, the stitching was ultimately accomplished.

Three days later three quarters of a pint of odourless clear fluid, which had accumulated since the first operation, were drawn out of the right pleura, and an opening was made into the lung; but though fœtid pus in small quantity escaped, no genuine cavity was opened. A similar deeper puncture into the lung three weeks later was not more successful, though a free communication with a bronchus was established. A certain amount of fœtid discharge escaped through the opening, but the expectoration soon became as profuse as before. The child was then taken away into the country, and before long the wound healed. Nothing of importance happened until April, 1889, ten months after my operation, when during a fit of coughing he brought up a portion of the vertebra of a rabbit. I should say that before he went away his father recalled most distinctly that he himself had been feeding his son with some mulligatawny soup on December 10th, 1887, that is seven months before I saw him. He recollected the choking fit, and his wife running in to see what was the matter, but curiously enough he had never associated it with the attack of supposed whooping-cough which began about this time, and was supposed to have been the cause of all the trouble. Some days after the expulsion of the bone the child was still coughing very much, and bringing up four ounces of very fœtid expectoration in the day.

The return to health was very gradual, and is even now not complete. In March of this year I heard from his father that the boy was the picture of health, but had not quite lost his cough.

In September he caught a cold, and has been more or

less of an invalid ever since. There was increased expectoration, and on a few occasions some slight hæmoptysis. I saw him on December 19th, 1895, and found him a well-grown lad, bright and intelligent; he is fat —rather too fat, indeed, and there is some clubbing of the fingers. The right side is retracted, and moves less than the other. The physical signs, as shown on the



accompanying diagram, point pretty clearly to the existence of a small bronchiectatic cavity at the situation of the

original operation. He brings up now about an ounce of almost inodorous pus, perhaps more, mixed with saliva, in the day, emptying himself two or three times. There is at present no fever, and I am assured there is no albuminuria. It is a question not very easy to answer whether it is wisest to leave him alone, or to endeavour, by incision and drainage, to secure the contraction of the cavity we think we know of. I believe the latter would be the best course to pursue, though the possibility of the existence of other cavities which could not be drained by the incision must not be forgotten.

Case 4.—W. H—, æt. 17, was admitted to the Brompton Hospital in May, 1885, under Dr. Powell; he said he had swallowed a straw some time before, which was followed by the spitting of a little blood. His illness began five months before admission with a hard dry cough, which soon disabled him. On admission he had a good deal of pain and some contraction of the right side of the chest, which was dull below the angle of the scapula behind, and the fifth rib in front, with absence of vocal fremitus and resonance, and weak breathing. The rest of the chest, except for a few scattered râles, was normal. There were from 14 to 16 oz. of frothy, stinking, mucopurulent expectoration, sometimes tinged with blood. The temperature varied from normal to 103°.

An incision in the fifth interspace evacuated a quantity of offensive pus. The finger passed into an irregular cavity with loose friable walls, which felt like broken-down lung tissue. The operation relieved him for a time, but the expectoration, though diminished, did not cease. Some burrowing of pus occurred, and rather more than a month after the operation he died of an abscess in the right occipital lobe of the brain. The right ear was healthy.

The lower part of the left lung, which was adherent to the chest walls, contained an irregular cavity, which passed downwards between the diaphragm and the ribs, and had actually perforated the diaphragm. In the

bronchus of the lower lobe there was part of an ear of corn with the stalk upwards, lying loose in the cavity of the tube, which at this point showed no change except a dark reddening of the lining membrane. A short distance below, however, there was a spot of ulceration at a point where the foreign body had originally lain. The bronchi in the lower lobe were all much dilated, the intervening lung tissue being collapsed, airless, and somewhat indurated. In the middle lobe was a thin-walled cavity as large as a pullet's egg, containing some pus (which did not contain tubercle bacilli). The bronchus leading to this cavity was only slightly dilated. The rest of the right lung and the left lung were healthy.

Another case is fully reported in the 'Lancet,' 1887, vol. i, p. 714. It was that of a man who had a tooth impacted in a branch of the right bronchus. He developed bronchiectasis, and was submitted to several operations by the late Mr. Marshall and myself. We did not discover the foreign body, and more than three years after the accident he died with tuberculosis engrafted on his previously diseased lung.

The points I am anxious to bring out in recording these cases are the following:

1. *Pathology.*—It appears that, when a foreign body becomes impacted in a bronchus and remains there, sooner or later the lung below the point of impaction will pass into a state of bronchiectasis. This will occur more rapidly if the foreign body is sharp and irritating, such as a piece of grass or an ear of wheat; more slowly if it be smooth, such as a tooth. In the latter case the lung itself will be firm and fibrous; in the former case a gangrenous and burrowing cavity is more likely to result. The process appears to result from the accumulation of septic discharges in the bronchi, the pneumonic changes being secondary, and it corresponds to that which occurs after the obstruction of a bronchus from the external pressure of an aneurism or other tumour, or the bursting

into a bronchus of any cavity containing septic material, such as a cancer of the œsophagus. I think that whenever a case of unilateral bronchiectasis presents itself, if none of the other recognised causes of the disorder appears to have been present, we should always suspect the presence of a foreign body. Of course it does not follow that the mischief may not ultimately extend to the opposite lung, but this appears only to take place after a very long time. I would also add that we should not reject, as probably apocryphal, an account of the inhalation of a foreign body given spontaneously by the patient; and also that, if such an account be not at first forthcoming, we should not give up the idea without a very searching cross-examination. For, while in some cases the accident leaves a most indelible impression on the mind, in others it seems to have caused an almost inappreciable amount of inconvenience at the time, and to have quickly faded from the memory.

2. *Prognosis.*—It appears that if the foreign body is rapidly expelled or extracted, or if it makes its way, as sometimes happens with sharp objects, to the surface, pretty considerable bronchiectasis may be recovered from, or at all events may cease to give rise to further trouble. But it must by no means be assumed that such a favorable result will occur. A comparatively short residence of a foreign body in a bronchus may set up so much mischief that the condition may be as unamenable to treatment as is a bronchiectasis arising from a pleurisy or any other cause; and it is easy to understand that this may be the case, seeing that the cavities are probably produced chiefly by the contraction of the surrounding lung tissue, and that the removal of the foreign body is only likely to affect favorably the mucous membrane lining them. The prognosis in these cases is therefore always doubtful and usually bad, and the possibility of the engrafting of tubercle on to bronchiectasis must not be forgotten.

3. *Treatment.*—The most important rule is that, if there

be anything like reasonable grounds for supposing that a foreign body has passed into a bronchus, no time should be lost in endeavouring to remove it. The inconvenience of an early tracheotomy and the resort to the usual methods of search (which I need not here dwell upon) are not for a moment to be weighed against the possibly irremediable damage that may be done by even a few weeks' residence of a foreign body in a bronchus. But if the mischief have arisen, an attempt should be made to reach the foreign body, even though the search may not improbably prove unsuccessful, and the probability of complete cure is small.

An attempt must be made to ascertain the position of the largest cavity by means of auscultation, and then it should, if possible, be opened and explored. Most likely adhesions will not be met with; it is therefore advisable to stitch the lung to the costal pleura before opening it, after the removal of a portion of rib, which in such a case is essential. It must, however, be remembered that the foreign body will most likely be impacted above the level of any cavity sufficiently large to give rise to any characteristic physical signs. A knowledge of the size of the foreign body may possibly enable the surgeon to guess whether it is likely to have stuck in the course of the main bronchus, or whether it may have passed into any other part of the lung.

A drainage-tube ought certainly to be placed in the wound, not only for the sake of enabling the cavity to contract, but also because the foreign body, if not discovered at the operation, may make its way out some time after the opening of the abscess. If it be not expelled, tracheotomy should be done, and the bronchi should be searched in the usual way, not omitting the plan of inverting and shaking the patient, and in suitable cases an incision should be made into some other part of the pleura than that through which the cavity was opened, and the surface of the lung should be explored with the finger.

In short, the condition being one which, if unrelieved, is almost certain to prove fatal in time, no method should be left untried which appears to offer the slightest rational prospect of success.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. viii, p. 167.)

NOTE ON THE ENDEMIC FEVER OF THE MEDITERRANEAN

BY

SURGEON-CAPTAIN M. LOUIS HUGHES
ARMY MEDICAL STAFF, MALTA

COMMUNICATED BY DR. GEORGE THIN.

Received December 23rd, 1895—Read April 14th, 1896.

IN the countries bordering the Mediterranean Sea south of latitude 46° N. (isotherm 55° F.), an endemic fever prevails, to which neither satisfactory name nor a place in the official nomenclature of diseases has yet been given. In the navy this disease is returned as "remittent fever," though it has no connection with fevers of malarial origin. In the army it is returned, together with all cases of slight and indefinite pyrexia, as "simple continued fever," in spite of its many and obvious distinguishing characters. A certain slight resemblance to enteric and malarial fevers has been used as an excuse for calling it by many hybrid and unscientific names, such as pseudo-tifo, adeno-tifo, typho-malarial fever, &c. Various other names have been given to it, none of which can be said to be satisfactory, such as febris complicata, febris sudoralis, pythogenic septicæmia, and such local names as Gibraltar fever, Rock fever, Malta fever,

Mediterranean fever. In this paper I follow the example of others, and call it "Mediterranean fever," with a hope that a more suitable name may be soon adopted.

The specific nature of Mediterranean fever is proved by its long and indefinite duration, irregular course, tendency to relapse, and peculiar symptoms; by the absence of intestinal ulceration, and by the constant presence in certain tissues of a definite species of micro-organism capable of producing by inoculation a similar form of pyrexia in monkeys.

The fever prevails mostly in the large seaports, but is also to be met with in inland villages, even at high altitudes. During the early part of the present century it appears to have existed both in Malta and Gibraltar, but to have been returned under such symptomatic headings as rheumatism, bronchial catarrh, lobular pneumonia, climatic cachexia, &c. The first accurate description published was that by Dr. Marston in 1861, written while serving as an army surgeon in Malta.[1] He was followed by Chartres (1865), Boileau (1866),[2] Oswald Wood, Notter,[3] and Donaldson[4] in 1876, also army surgeons serving in Malta. In 1879 Prof. Veale,[5] and in 1885 Prof. Maclean[6] published valuable reports dealing with cases invalided to Netley from the Mediterranean. In 1887 Surgeon-Major Bruce published his discovery of a causal micro-organism, and later on other papers;[7] while

[1] 'Army Medical Department: Statistical, Sanitary, and Medical Reports,' iii, 1861 (published in 1863), p. 486.

[2] Ibid., 1865 (published in 1867), p. 527; 1866 (published in 1868), p. 478.

[3] 'Edinb. Med. Journ.' xxii, 1876-7, pp. 40, 289.

[4] 'Army Medical Department: Statistical, Sanitary, and Medical Reports' for 1876 (published in 1879), p. 238.

[5] Ibid., 1879 (published in 1881), p. 260.

[6] 'Practitioner,' xxxiv, 1885, p. 78; and 'Brit. Med. Journ.,' 1875, vol. ii, p. 224.

[7] 'Practitioner,' xxxix, 1887, p. 161; and xl, 1888, p. 241; 'Annales de l'Institut Pasteur,' vii, 1893, p. 289; articles in Davidson's 'Hygiene and Diseases of Warm Climates,' p. 265, 1893, and Quain's 'Dict. of Med.,' vol. ii, p. 10, etc.

Surgeon-Captain Moffat in 1889 dealt with its occurrence in Gibraltar.[1]

Since that date I myself and others have endeavoured to carry on these investigations.[2] In Italy and Sicily, Borrelli (1872), Tomasi (1874), Cantani (1878), Rummo (1881), Galassi (1883), Capossi (1885), Federici (1885), Tomaselli (1886), Guiffre and Silva (1893), and many others have published papers;[3] as have also Carageorgiades of Cyprus (1891), and Typhaldos of Greece. These and others have met with the fever as an endemic disease in Middle and Lower Italy, Sicily, Constantinople, Crete, Greece, Smyrna, Cyprus, Malta, Gibraltar, Tunis, Algiers, &c.; while Drs. Hutchinson Miles, Pasquale, and Rho[4] would lead us to believe that the same fever exists on the borders of the Red Sea. As in India and elsewhere there still exist forms of remittent fever of long duration and irregular course, neither enteric nor malarial in nature, it would be unwise to lay down any geographical limits to its prevalence in the limited state of our present knowledge.[5]

Symptomatology.

So variable are the symptoms, and so uncertain is the duration of this fever, that it is impossible to give a description to which all cases can be referred. Putting aside those short (abortive ?) and indefinite cases of fever lasting from seven to twenty-one days, which are more severe than simple ardent fever, and unlike enteric fever,

[1] 'Army Medical Department: Statistical, Sanitary, and Medical Reports,' xxxi, 1889 (published in 1891), p. 403.

[2] 'Lancet,' ii, 1892, p. 1265; 'Annales de l'Institut Pasteur,' vii, 1893, p. 628.

[3] 'Trattato di Medicina' di Charcot, Bouchard, and Brissaud, 22a del., vol. i.

[4] 'Lancet,' i, 1892, p. 1359; 'Sperimentale,' anno xlviii, p. 545, etc.

[5] The writer hopes shortly to publish a complete bibliography of this fever.

we meet with three varieties, to which all cases of this Mediterranean fever seem to approximate clinically. The following descriptions are based on notes of over a thousand cases which I have had the opportunity of seeing in Malta during five years of constant hospital practice.

1. *Malignant or fatal type.*—(*a*) The patient, often a strong muscular man, is admitted to hospital suffering from severe pyrexia, stated to be of only one or two days' standing. He complains of severe headache, "pains all over him," distaste for food, possibly nausea, and even vomiting. His face is flushed, his tongue thickly coated with whitish-grey or yellow fur, pink at the tip and edges, moist, swollen and indented by the teeth laterally. The temperature is 104°—105° F. There is epigastric, splenic, and perhaps hepatic tenderness on pressure, while the area of splenic dulness is increased. Diarrhœa may be present, when it is usually due to involvement of the large gut. If left to take its course, after four or five days of constantly high pyrexia, signs of basal pneumonic congestion, with bronchial râles all over the chest, appear, and these may pass into lobular consolidation. The pulse remains strong and the mind clear. The urine is decreased in amount, dark in colour, and loaded with lithates; diarrhœa, if present, becomes profuse and frequent, the stools being usually brown in colour, very offensive, but variable in consistency. After a varying number of days the pulse begins to flag and become intermittent, the breathing laboured, and obstinate vomiting may be present. The tongue becomes brown, the teeth are covered with sordes, and other symptoms of the so-called "typhoid state" set in; the respirations are shallow and fast; delirium supervenes and passes into coma; the fæces are passed involuntarily; the temperature rises, the heart gives way, and the patient dies of hyperpyrexia. At death the temperature is, as a rule, about 110° F., but may continue to rise, so that a temperature of 112°—115° F. has been registered shortly after in the internal organs. Rapidly fatal cases are rare in

these days of improved treatment. In a few cases there is a temporary fall to normal with great exhaustion, for a few hours before death from hyperpyrexia; while other delicate individuals appear to die of the direct toxic effects of the virus, at a temperature well below hyperpyrexia.

(*b*) In some cases the patient passes safely through the primary attack, the pyrexia abates at the end of two or three weeks for a few days, followed by a gradual rise and relapse, in the course of which similar fatal symptoms may supervene.

(*c*) In cases which prove fatal at a later stage in the disease (70th to 150th day) death is usually due to sudden cardiac failure, to debility and exhaustion, or to the supervention of phthisis or some other intercurrent disease.

Of twenty-nine fatal cases, death occurred in 13·7 per cent. during the first week in hospital, in 18·3 per cent. during the second week, in 25 per cent. during the third week; in 57 per cent. during the first month, 22·6 per cent. during the second month, 9 per cent. during the third month, 4·5 per cent. during the fourth month, 2·2 per cent. during the sixth month.

2. *Undulatory type.*—These cases are marked by intermittent waves of remittent pyrexia of variable length, marking the pyrexial intensity, separated from one another by periods of temporary abatement or absence of symptoms. In typical uncomplicated cases, confined to bed, there is usually a premonitory stage of low spirits, sleeplessness, anorexia with dyspeptic symptoms, and each evening headache and and slight pyrexia. The temperature next rises gradually, remitting each morning about half the amount of the previous evening rise. With this are combined slight headache, pains in the back and limbs, moist, furred, swollen tongue, a bad taste in the mouth, epigastric tenderness, and constipation. Most patients, however, do not at first report themselves sick, but, imagining that they have "a bilious attack" or "liver," take aperient pills, and attempt to work the

illness off by hard exercise. The result is that all the symptoms become exaggerated, and on admission the temperature is high, and the headache severe. The temperature having reached 103°—105° F. in the evening, accompanied by some bronchial catarrh or hypostatic pneumonic congestion in proportion to the severity of the case, after a variable period gradually falls to normal or thereabouts in the morning; and though it may be slightly higher in the evening, the patient feels better and wants to get up, while the primary wave may be said to be over. After a day or two, however, the temperature again begins to rise, and a relapse ensues, similar to the primary attack, but usually less prolonged and less severe. This subsides, but is followed by other relapses forming the undulatory temperature charts so characteristic of this fever. Such pyrexia is nearly always accompanied by obstinate constipation, though diarrhœa may occur temporarily in very severe cases, more especially during the primary attack. Each daily remission of temperature is accompanied by profuse sweating. Anæmia and muscular wasting are progressive and often extreme. At any stage, but usually late in the attack or during convalescence, symptoms of localised interstitial neuritis may occur, leading to obstinate sciatica, intercostal neuralgia, &c., or to symptoms referable to irritation of the peripheral sensory nerves or of the nerves of special sense. In many cases effusion into one or more joints may suddenly occur, of a transitory and metastatic nature, but causing extreme tension and pain; or painful orchitis may appear. Finally the patient is reduced to an emaciated, anæmic, bedridden condition; subject to attacks of bronchial catarrh, lobular pneumonia, cardiac palpitation, rheumatic or neuralgic complications on the slightest exposure to chill, change in the weather, or excitement. Disappointed at each relapse, his whole expression is the picture of despondent apathy, his only wish to get away to England. His emaciated appearance, his profuse night sweats, often

intermittent pyrexia and cough, remind one forcibly of the late stages of phthisis. Gradually, however, towards the end of the second or third month, his temperature becomes normal or subnormal in the morning, and but slightly above normal in the evening; next the evening rise ceases, giving place usually to a day or two of subnormal temperature, after which convalescence is established. The strength now slowly returns, the cheeks fill out, and the patient gets up for an increasing period every day. After a few weeks, if not invalided home, he is sent to a sanitarium, or perhaps to duty, but for months after is liable to attacks of neuralgic pain, to swollen joints or testicles, combined with slight pyrexia. Final recovery cannot be said to take place for many months, and until the anæmia has disappeared and the mucous membranes have regained their complete functions; when once, however, completely free from the disease, it does not recur like paludism.

The average duration of pyrexial symptoms is sixty to seventy days, but cases have been known to last thrice as long. The average stay in hospital is over ninety days, and convalescence has been prolonged to a year or more in certain cases.

3. *Intermittent type.*—In these cases the temperature intermits daily, and being of a non-paroxysmal nature, they resemble hectic fever cases. If, however, the temperature be accurately taken, so as to register the daily maximum and minimum, these are found to form waves of pyrexial intensity similar to those previously described, the daily remittance being exaggerated into an intermittence. These cases are, as a general rule, shorter than the undulatory ones, complications being milder in character when present. Constipation, night sweats, and progressive anæmia accompany the pyrexia, while arthritic effusion may occur. The patient is usually less despondent, the absence of marked undulations does away with the disappointment felt at each relapse, the patient's daily remark being that he is "just the same." In many

cases non-paroxysmal pyrexia and profuse night sweats are the only symptoms present, and the patient, if allowed, wishes to get up and eat solid food each morning, but suffers from slight malaise in the afternoon. Such cases go on steadily in spite of all the drugs in the pharmacopœia, and though an indiscretion may bring on serious symptoms, they seem to cease spontaneously, or at the commencement of some new line of treatment which, however, fails to stop other and similar cases.

Between the undulatory and intermittent types we meet with every variety of curve that can be said to approximate to the above individual descriptions, or to a mixture of both; but there is always a tendency to the formation of waves of pyrexial intensity if the curve is accurately registered.

Special symptoms.—In severe cases the face may be cyanosed, but in long cases towards the end of an attack the face becomes of a dull clay colour, the skin tightly drawn over the skull, with an expression of listless resignation to an uncertain fate. There is no exanthem, but sudamina are not uncommon during and after the third week, especially when the skin is not properly attended to. Prickly heat (which may become pustular) is an annoying complication of hot weather, while in the spring and autumn boils may occur. Subcutaneous hæmorrhages, combined with scorbutic symptoms, are of rare occurrence. About the fourth week desquamation takes place, being most noticeable on the soles of the feet, where the skin peels off in large flakes. Towards the end of long attacks the hair falls out extensively, but is gradually replaced by new growth during convalescence. As in enteric fever, there is a certain amount of cutaneous bronzing, but nothing approaching the pigmentation of paludism. A distinctive and disagreeable odour is present in nearly all cases, being most especially marked at post-mortem examinations. The profuse diaphoresis following the fall of temperature when intermittence is present is most characteristic, and gives rise to the name "febris sudo-

ralis." The sweat rolls off the patient's face in large drops, soaking through the pillow; while at the same time it soaks through his flannel suit, the sheets, and even the blankets, occurring usually about one or two o'clock in the morning; this lasts an hour or more, necessitating two or three changes of clothing.

Pyrexia is the chief and often sole symptom present, and although, speaking generally, there is a relationship between the temperature curve and the other symptoms present, yet the pyrexial severity is not always an indication of the urgency of the symptoms or of the prognosis in any given case. Its height in a large number of cases seems to depend upon the capacity of the individual to nervous excitability.

The chief characteristic of the pyrexia of this fever when compared with that of others, is the variability which exists in its amount and duration in different cases. The daily curve may vary between a continuously high temperature and an intermittent one. One point, however, is common to all cases, in that the daily maximum and minimum temperatures tend to form waves of intensity of varying character and duration. These waves in individual cases have a tendency to resemble their primary wave, though they usually decrease in length and severity as the cases progress.

The typical wave in uncomplicated cases confined to bed rises like the ideal curve of enteric fever, and subsides in an equally regular manner (see Charts II and VII). Generally speaking, however, all sorts of variations occur (see Charts I, IV, VII, and XII), even to a sudden fall from a continuously high temperature to normal. The latter fall is not uncommon as a temporary abatement (see Chart X), but has only occurred three times in the writer's experience as a sudden permanent recovery by crisis. Not infrequently permanent cessation of pyrexia is immediately preceded by a burst of unusual severity (see Chart V), while convalescence is almost always preceded by a period of subnormal temperature

lasting from one to three or more days (see Charts I, V, and VII). The average length of 300 well-defined waves was about ten days, the primary wave being usually longer (eighteen to twenty-three days), or it may even last almost the whole pyrexial period (see Chart III, where it lasts fifty-four days). The durations of these 300 waves, occurring in ninety-five undulatory cases, were—

One week and under	70
Two weeks	97
Three „	60
Four „	23
Five „	42
Six „	3
Seven „	3
Eight „	2
	300

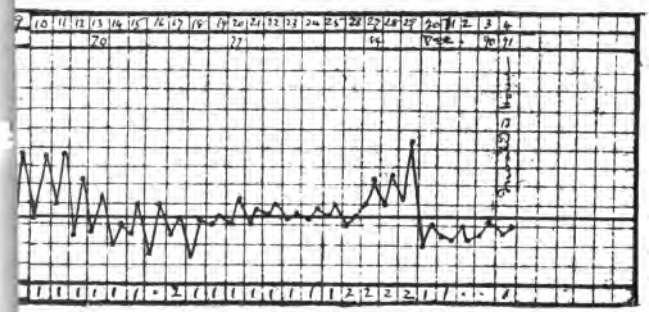
The average number of waves in an attack was three (one to seven). The interval between waves is marked by a period of apyrexia, without other morbid condition, lasting from one to ten or even more days (average three to four), or simply by a comparative abatement of pyrexial and other symptoms of variable duration and degree.

The daily temperature curve is usually remittent in character, the morning temperature being one or two degrees lower than in the evening, without actually touching normal except during the intervals between waves of pyrexia. It may, however, assume in different cases any type between intermittence and a continually high temperature, or pass from one type to another in the same attack (see Chart VII).

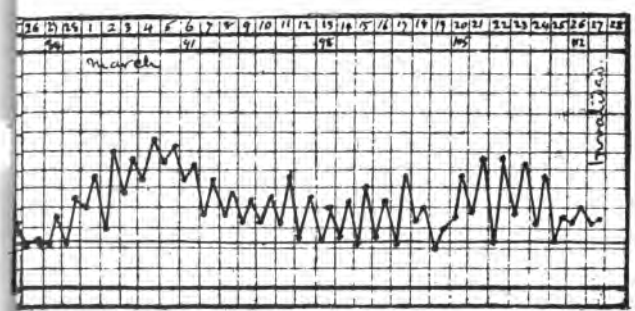
In intermittent cases (Chart VI) the temperature is usually at or near normal during the morning visit (8 a.m.), and begins to rise steadily a little before 11 a.m. to a maximum between 2 and 3 p.m. From this it gradually falls to halfway about 6 p.m., and reaches normal again about 10 or 11 p.m. or later, after which

See Explanation of Charts, page 254.

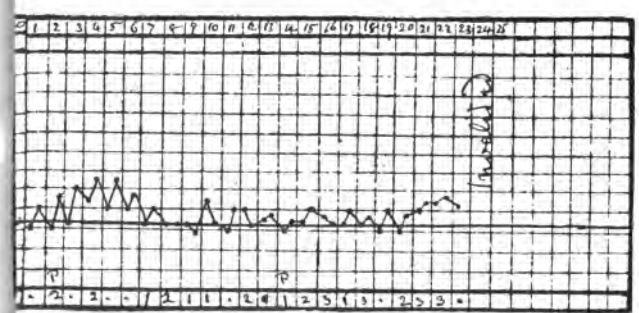
1



2



3



4

profuse diaphoresis occurs. Occasionally slight supernumerary rises of temperature are added to this curve, a common one being just after the evening visit and bed-making. This curve is accentuated by mental emotion or excitement, by exertion, injudicious dietary or medicine, excessive constipation or the reverse, by the damp enervating sirocco wind, or by the appearance of any localised special symptom. The relief of constipation or the cessation of a sirocco wind often proves an excellent antipyretic. These maximum and minimum may be postponed, so that in a few extreme cases the morning temperature becomes the high one, and the afternoon the reverse. This daily curve is non-paroxysmal, resembling more the hectic fever of phthisis than the paroxysms of ague, the daily rise being associated with a feeling of chilliness and malaise only, rigor being absent except in a few cases of specially nervous temperament.

From the above mild type, the daily temperature curve may increase in duration so as to postpone the fall well into the night, and the diaphoresis to the early morning, while at the same time the morning rise is anticipated. In more severe cases one curve appears to run into and overlap the next, until with less and less remittence the temperature comes to be continuously high, the daily variation being less than one degree, a condition leading to danger from sudden hyperpyrexia.

The pulse is usually firm and slow at first (80—90), even out of proportion to the number of the respirations, and the amount of pyrexia present. In malignant cases, where there is lung stasis, it is rapid, and becomes small, thready, and then intermittent before the overburdened heart gives out entirely. In long-continued cases it often becomes constantly increased in rate (110—120). In such cases cardiac irritability is of common occurrence, giving rise to attacks of palpitation on the slightest exertion, or even under the influence of some trifling emotion. Hæmic murmurs are met with during convalescence. Organic cardiac disease is said to arise in some cases, but I

have only met with it in four instances (fatal on the 19th, 62nd, 111th, and 150th days), and in these the condition might have existed previously. In the first two cases the immediate cause of death was pericardial effusion (similar in onset to the characteristic arthritic effusions). Swelling and œdema of the ankles after standing is common during convalescence. The writer has only once met with phlegmasia dolens following an attack of this fever. The blood has been microscopically examined by Dr. Thin and numerous other observers, and culture experiments have been made, but no organisms have been found present. The spleen can nearly always be made out on percussion and palpation, and occasionally is considerably enlarged. During the first acute stage it is tender to pressure, and may be painful. The specific micro-organism has been isolated from the spleen during life. Epistaxis occasionally occurs early in an attack; intestinal hæmorrhage is limited to spots of fresh blood in the stools in cases where the lower bowel is affected.

About the beginning of the third week, or earlier in severe cases, bronchial râles may be heard on auscultation in nearly 95 per cent. of cases. In acute cases basal congestion of the lungs is a most common symptom. In severe cases, and especially in those who have previously suffered from pleurisy, pneumonia, or have organic heart lesions, this is apt to pass on to double lobular pneumonia of varying amount, most marked, as a rule, on the right side. A nervous cough unaccompanied by expectoration is occasionally present; while in mild intermittent cases emaciation and night sweats may be combined with bronchial râles and crepitations, and give rise to a wrong diagnosis of phthisis. Pleuritic effusion without suppuration is not uncommon, and often leaves permanent adhesions behind.

The tongue is usually thickly coated with whitish-yellow fur on the dorsum, pink at the tip and edges, moist, swollen, flabby, and indented laterally by the teeth. In very severe cases only does it tend to become dry and

brown. Occasionally it becomes red, glazed, raw, and with the epithelium denuded in patches. There is usually foulness of breath, tenderness on pressure in the epigastric region, nausea, and occasionally vomiting, with other signs of gastric derangement. In non-malignant cases constipation is the rule; in my experience constipation has been marked in 81 per cent., diarrhœa in 4 per cent., both conditions in 3 per cent., and a normal condition in 12 per cent. of such cases. In fatal cases, owing to the frequent involvement of the lower bowel, diarrhœa has been present in some 50 per cent. of those that were noted. The condition of the tongue is a valuable indication of the suitability of the diet given, and of the permanency of any amelioration of symptoms. A fall in the temperature rarely proves permanent if the tongue remains coated.

Albuminuria is rare even in fatal cases, though a form of large white kidney has been met with in very prolonged cases.

The action of the virus on the nervous system may be regarded as one of the special characteristics of the fever, of which indeed many of the symptoms already mentioned may be more or less the result. Severe headache with shifting pains in the back and limbs is rarely absent in the initial stages. Later on constant or repeated attacks of facial or occipital neuralgia may be present. In a large number of cases, generally late in the attack, or even during convalescence when pyrexia has ceased, other nerves may be affected, and obstinate lumbago, intercostal neuralgia, or sciatica may be set up. In rare cases the acute stage is accompanied by general cerebro-spinal irritation, characterised by mental irritability, delusions, sleeplessness, cutaneous hyperæsthesia of variable extent, girdle pains, &c. Severe pain and hyperæsthesia of the soles of the feet is a not uncommon condition. Paralysis, partial or complete, of certain muscles is a late symptom. The extensors of the foot and the deltoid are most commonly affected. The muscle slowly atrophies, and as slowly regains, first its function and then its proportions,

recovery being complete. The special senses of hearing and touch become temporarily diminished in a number of cases without the administration of quinine. Taste is usually impaired, vision to a small extent occasionally. The decubitus is lateral until the patient becomes dangerously ill. Delirium is not common except in very severe cases, when it may pass into coma before a fatal issue. The writer has not met with mania or imbecility following attacks of this fever. The mind is clearer at the beginning than in enteric fever, but during convalescence the power of concentration of thought and of remembering names or figures is often temporarily impaired. Wakefulness and sleeplessness are common at first, and the nervous prostration following an attack is most marked. Retention of urine may occur during the acute stages. The effect on the mechanisms of heat regulation have been mentioned.

Effusion into one or more joints is a very common and characteristic symptom, which may occur during the acute stage, but is more common during the third week or later. In some cases this condition, combined with very slight pyrexia, may be the only symptom complained of on admission, leading to a first diagnosis of rheumatism or even synovitis. The joints are attacked in something like the following frequency :—Hip, knee, shoulder, ankle, wrist, fingers, toes, elbows, sacro-iliac synchondroses, intervertebral joints and lower jaw, &c. This may come on suddenly in the course of an hour or two, and may disappear in a few hours or days, only to be replaced by a similar affection in some other joint, and this may be repeated over and over again, usually finishing up with the fingers or toes, having no regard to symmetry. The joint rapidly fills up, with great pain and occasionally some redness. Neither suppuration nor ankylosis occurs, but if the condition persists for long some œdema may be present in the surrounding tissues, causing it to resemble a severe gonorrhœal joint. Pain and stiffness in the aponeuroses or muscle sheaths may occur.

These symptoms appear to have a special predilection for those who have previously suffered from rheumatism or rheumatic fever elsewhere.

The rheumatic and neuralgic symptoms have often a relation to chills received during an attack of the fever.

Epididymitis and orchitis (usually single) occur at a late stage in a few cases, mastitis very rarely. The testicle swells in from twenty-four to forty-eight hours to the size of an orange, is extremely painful, while there may be some redness of the skin and effusion into the tunica vaginalis. It is often long in disappearing.

Abscesses occasionally occur, but are probably unconnected with the disease in question.

Ætiology.

(*a*) *Bacteriology.*—Although the disease has existed in the Mediterranean for so many years, it was not until 1886 that a micro-organism was discovered in Malta by Surgeon-Major Bruce (recently Assistant Professor of Pathology in the Army Medical School, Netley), to the effects of which the phenomena of this fever could be attributed. Bruce's first case died on the fifteenth day of the disease, when he found nine hours after death, in splenic sections, "enormous numbers of micrococci" scattered through the tissues. In May, 1887, with Dr. Caruana Secluna, he made between thirty and forty inoculations into sterilised agar-agar with blood aseptically taken from the fingers of ten cases of this fever without result. One of these cases, however, proving fatal, eight tubes of agar-agar were inoculated with the usual precautions at 6.30 p.m., less than one hour after death. These were kept at the temperature of the air (25° C.) until 11 a.m. the following morning, when six were placed in an incubator at 37° C., while two remained at the temperature of the air. Colonies of a micro-organism, which the writer has named the *Micrococcus miletensis*, appeared in all these tubes. In the

next case Bruce was unable to make an autopsy, but seven hours after death, by means of a sterilised trocar and cannula, he obtained some splenic pulp, with which he inoculated six tubes; these being placed under similar circumstances produced similar growths. These, with six other cases in which he obtained the micro-organism from the spleen after death, and one case in which he obtained it from the spleen during life, making ten cases in all, have all been published in the 'Annales de l'Institut Pasteur' and elsewhere with full details.

Surgeon Gipps, R.N., describes two cases in the 'Transactions of the Epidemiological Society,' vol. ix, in which he isolated a micrococcus, of which he gives drawings. Though his work was done in Bruce's laboratory he does not appear to place much confidence in its causal connection with the disease.

I worked for a short time with Bruce, and have isolated from the following thirteen cases a similar micro-organism, the fullest precautions being taken to eliminate error.

Case 1.—Admitted July 27th, 1891, æt. 22. Service two years, previous health good. Admitted with the usual febrile symptoms, onset being sudden. On the tenth day the symptoms partook of a typhoid character, the tongue became dry and brown, the pulse quick and feeble, the abdomen tympanitic; there was also subsultus tendinum, and abundant crops of sudamina. This continued until the twenty-fourth day, when he began to improve, the disease being characterised by constipation and rheumatic symptoms to the end, whereas from the thirteenth to the twenty-sixth day there had been a tendency to diarrhœa. From the fortieth to the forty-fifth day there was an apyrexial period, after which there was a severe relapse. The condition gradually became worse, tympanites set in, and was a prominent symptom. There were signs of hypostatic congestion at the bases of the lungs at the back. He died much exhausted at 6.30 a.m. on the

seventy-third day of the disease. The pyrexial curve was similar to that of Case 2, except that the first or primary wave lasted thirty-nine, and the second (fatal) wave twenty-seven days.

Treatment.—No solid food, stimulants, antipyrin, salicylate of soda, diaphoretics, sponging, &c. Quinine in various doses from two to fifteen grains three times a day.

Though not in clinical charge of the case, I had constant opportunities of visiting the patient during life, and there was no doubt as to the diagnosis of Mediterranean fever. At the post-mortem examination four hours after death, six army and three civilian medical officers were present, who all confirmed the diagnosis. The body was fairly well nourished, and the heart normal. The lungs showed some hypostatic congestion of both bases, with sero-purulent exudation in the bronchioles. Liver weighed 74 ounces, was slightly fatty. Kidneys normal. Spleen 12 ounces, enlarged and dark, but firm in texture. Stomach and duodenum normal; the jejunum had a patch of congestion, four and a half inches long, a foot below the duodenum. The ileum, Peyer's patches, and the solitary glands were all normal. In the large intestine the cæcum was normal, but just below this for one foot in extent, and again for six inches at the upper part of the sigmoid flexure, were patches of congestion and exudation. There was no sign of ulceration along the whole extent of the alimentary tract.

Experiment.—The spleen was removed as aseptically as possible, and without tearing its capsule; at once wrapt in a towel soaked in a solution of perchloride of mercury, and removed to the laboratory. There three cuts were made in the spleen with three sterilised knives, each cut being through, and at right angles to the plane of the preceding one, the innermost cut being used for inoculation purposes, the cuts being allowed to fall together between each operation. Three tubes of agar-agar were inoculated by a small drop of blood removed on a sterilised platinum ooze, two tubes were at once placed in the incubator at

99° F., and one left at the temperature of the air (about 75° F.). Characteristic colonies of the *Micrococcus miletensis* appeared in both the tubes placed in the incubator after 124 hours, but the tube left at the temperature of the air remained sterile. These growths were passed through six generations of pure cultures on agar-agar, and were used for inoculation of monkeys. One primary and two secondary cultures forwarded to Bruce at Netley were identified by him as the same micro-organism that he had previously found in such cases.

CASE 2.—Similar to the last in character. Admitted July 10th, 1892. Age 20; service six years, two of which had been in Malta. This man slept in a bed from which his comrades in his room said that other cases had been admitted to hospital. The bed in question was under a roof ventilator, placed next a main drain ventilator, the smell from which was complained of in the room.

Previous history.—Had suffered from slight attacks of febricula and bronchial catarrh in Cairo in 1887; from gonorrhœa four times in Malta between October, 1891, and June, 1892. Habits latterly intemperate, physique on admittance fairly good.

Clinical history.—Onset somewhat sudden, admitted on the third day of the attack with pains in the back and limbs, epigastric tenderness, and some vomiting, the last relieved by bismuth. Primary attack lasted twenty-four days, during which time there was a tendency to diarrhœa, but no other symptom of an enteric nature. After an apyrexial period of about five days the second wave began on the twenty-fifth day of the disease, the temperature remaining high, and the patient becoming daily worse. On the forty-ninth day he suffered from subcutaneous hæmorrhages over the sacrum and buttocks, but no bedsores appeared. There was tenderness on pressure in the epigastric, splenic, and left iliac regions, a tendency to diarrhœa with loose yellow stools throughout the relapse; while during the

last four days the stools were passed involuntarily. Death occurred on the fifty-fourth day at 7.15 p.m. from heart failure and exhaustion.

Treatment.—Careful dietary, and free use of stimulants. Gastric sedatives, antipyrin, cold sponging. Lead and opium internally, and opium and starch enemata for diarrhœa.

Examination one hour after death.—Body emaciated. Heart normal. Lungs showed hypostatic congestion of both bases. Spleen weighed 14 ounces, was very dark in colour, and soft in consistency. Liver weighed 59 ounces, was slightly congested. Stomach distended with gas and fluid food, but was otherwise normal. Duodenum normal. Small intestine contracted and shrunken, and about two and a half feet from the cæcum it was hyperæmic, with arborescent congestion in the course of the vessels. Peyer's patches and the mesenteric glands were all normal. The large intestine for two feet from the ileo-cæcal valve was intensely congested and offensive, which explained the presence of diarrhœa during life.

Experiment.—Cover-glass preparations of fresh splenic substance showed micrococci here and there. Three tubes of agar-agar were inoculated in the same manner as in the last case, and placed in the incubator at 99° F. Characteristic growths appeared in every tube within 120 hours of inoculation.

Case 3, a malignant one, proved fatal on the twenty-third day from hyperpyrexia. He was the only fatal case that occurred in the epidemic mentioned on page 239 (2). For temperature see Chart IX. At the post-mortem examination the lungs were found to be much congested at the bases; the spleen congested, friable, and weighing 15 ounces; the liver congested, but Peyer's patches and the mesenteric glands normal. Inoculations were made in broth and on agar-agar, characteristic colonies appearing on the seventh and sixth days respectively at 99° F.

Case 4, also a malignant case in a delicate subject, proved fatal from cardiac failure on the twelfth day. At the post-mortem there were mitral vegetations, with fatty degeneration and infiltration of the heart. The spleen weighed 13 ounces, and was extremely congested; liver enormously enlarged (88 ounces), but Peyer's patches and the mesenteric glands normal. The spleen was removed one hour after death, and three tubes of agar-agar inoculated, characteristic growths appearing on the fifth day at 99° F.

Case 5 died on the eighteenth day of continuously high pyrexia, in a comatose condition. At the post-mortem examination, seven hours after death, the lungs showed basal congestion; the liver (73 ounces) was congested and friable; the spleen (21 ounces) was almost in a state of liquefaction, its substance breaking up on the slightest pressure. The mesenteric glands were slightly enlarged, but Peyer's patches were normal. The great gut for eighteen inches, including the cæcum, was deeply congested, somewhat swollen and thickened, and the solitary glands were prominent. Characteristic growths were obtained from the spleen on agar-agar in five days.

Case 6 died of collapse, vomiting, and exhaustion on the thirty-fifth day of the disease, the temperature having fallen the day before death only to rise again. After death the bases of the lungs and the spleen (18 ounces) were found to be congested, the mesenteric glands slightly enlarged, but Peyer's patches and the intestines normal. Characteristic growths on agar-agar were obtained from the spleen in 130 hours at 99° F.

Case 7 died of heart failure and pericardial effusion on the nineteenth day of a very remittent case. After death the autopsy showed lobular consolidation of the lungs, the spleen (12 ounces) congested, the liver nutmeg, and the heart with mitral vegetations. Peyer's patches and the

mesenteric glands normal. The micro-organism was visible in cover-glass specimens of splenic substance, and was obtained from the spleen on agar-agar in pure growths on the sixth day.

The last two cases slept near a broken drain in a previously healthy barrack.

CASE 8 died of hyperpyrexia on the twenty-fourth day of continuously high pyrexia, after admission to hospital. After death there was basal congestion of both lungs, the spleen (12 ounces) was congested, the large gut much congested, the mesenteric glands slightly enlarged, but Peyer's patches normal. One hour after death the micro-organism was visible in cover-glass preparations of fresh splenic substance, and characteristic growths were obtained on agar-agar on the fifth day.

CASE 9, a long case of short undulations (see Chart IV), died of sudden hyperpyrexia on the fifty-seventh day of the disease. After death the lungs were œdematous and congested at the bases, the liver (80 ounces) intensely congested, spleen (11 ounces) congested but firm, mesenteric glands and Peyer's patches normal. Micrococci visible, and growths obtained as in the last case.

CASE 10, a somewhat unique one, was admitted to hospital for three weeks' fever and then discharged, apparently cured. Four months afterwards he was again admitted with fever of a typically intermittent character, which proved fatal from cardiac failure on the 154th day of the attack. After death the heart was flabby, there was a small aneurismal dilatation above the posterior left semilunar valve, and all three of these valves had vegetations on them. There was much serous fluid in the pleural cavities, the bases of the lungs being congested and œdematous. The liver (76 ounces) was nutmegged, the spleen (14 ounces) congested; but Peyer's patches, the

mesenteric glands and intestines were normal. Six tubes inoculated from the spleen all showed characteristic growths on the sixth day.

Case 11 died of hyperpyrexia on the twenty-fourth day of acute fever. After death there was considerable congestion of the bases of both lungs; the liver was nutmeg; the spleen (14 ounces) engorged with venous blood. There were patches of congestion here and there in the intestines, most marked in the colon. Peyer's patches and the mesenteric glands were normal. Characteristic colonies were obtained from the spleen as in the other cases.

Case 12 had served for three years in Gibraltar, and for five months in Malta. He died suddenly of cardiac failure, after a slight excitement on the 111th day of the disease. At the post-mortem examination warty vegetations were found on the mitral valve, there were recent pleuritic adhesions at the base and posterior parts of the right lung, the lower lobe of which was consolidated. The spleen (15·5 ounces) was congested, soft, and friable, the liver congested. The kidneys showed the characteristic appearances of "large white kidney." The intestinal walls were attenuated, but were otherwise healthy. The spleen was removed three hours after death, and from it characteristic growths were obtained, while sections were made of portions of the different organs.

Case 13 died somewhat suddenly on the sixty-second day of the disease, of effusion into the pericardium, during a relapse after his temperature had been normal for seventeen days. At the post-mortem examination 17 ounces of fluid were found in the pericardium, the right pleura was obliterated by organised lymph; the right lung œdematous and congested; the liver nutmeg; the spleen (15 ounces) congested, but the intestines, Peyer's patches, and the mesenteric glands normal.

Characteristic growths were obtained from the spleen on the fifth day after inoculation.

These altogether make thirteen cases in which I have successfully isolated the micrococcus from the spleen after death. If Bruce's ten cases and Gipps's two cases be added, we have twenty-five cases,[1] which make more than a coincidence.

The only case in which the growth was not found was in one published by Bruce. The failure was due to the use of too alkaline agar, a fact proved later by control experiments with growths from my cases on the same agar.

No other micro-organism has been found present under similar circumstances, nor has the writer met with a similar organism present in many other cases in Malta which he has tested, and which died from other causes.

Description of the Micro-organism.

The *Micrococcus miletensis* grows best on nutrient material, the alkalinity of which is slightly less than that of human blood, and at a temperature of from 37° to 38° C. At temperatures between 40° and 42° C. growth is suspended; above 42° C. artificial growths die. Below 18·5° C. growth is also suspended, while if they are kept at a moist temperature of 15·5° C. for long they die. Colonies that were allowed to dry completely were found to be dead when tested three years after. On the sloping surface of 1·5 per cent. peptone agar, at a temperature of 37° C., its colonies become visible to the naked eye in from 90 to 125 hours after primary inoculation from the human spleen. They first appear as minute transparent colourless drops on the surface of the agar, appearing somewhat like the agar itself. In about thirty-six hours more they become a transparent amber colour, and increasing very slowly in size, on the fourth or fifth day of their appearance become opaque. At this stage they

[1] I have since isolated this micro-organism from a further case, fatal on the 117th day of the disease.

resemble split pearls on the agar surface. Under a low power and with transmitted light such colonies appear to be orange in colour, quite round, with a definite but granular margin. If kept on moist agar they increase slowly in size, and while retaining their circular shape individually, may gradually coalesce. In the course of three months these colonies turn to a buff or even orange colour to the naked eye, and increase in thickness by heaping up material in the centre of the colony. The individual colonies never grow to any great size when compared with other growths, ceasing to grow when a little larger than a split pea. No liquefaction takes place. Though they do not increase in size after two months' growth, the colonies retain their vitality, at a suitable temperature, for over three months. The micrococcus will not grow on agar, the alkalinity of which is in excess of that of the human blood; but if cultivated on successive media of increasing alkalinity they can be educated to grow on very alkaline media. In this case, however, they are longer in appearing and grow more slowly in a very diffuse manner on the agar surface, with only abortive attempts at the formation of definite colonies. These diffuse growths, however, when transferred to agar having a suitable alkalinity, again revert to their original characteristic mode of growth. In making primary inoculations on old agar, the blood transferred from the spleen by drying, occasionally interferes with the growth of the colonies. This never occurs when the primary inoculation is made in broth, and secondary inoculations from this to agar made a few days later.

The micrococcus grows also in bouillon and gelatine. On the latter it grows very slowly at 22° C. without liquefaction. In the latter it gives rise to a general and increasing opaqueness, commencing on the fifth or sixth day, and afterwards forms a white precipitate consisting of these cocci, but without forming a surface pellicle.

Microscopically in the hanging drop they appear as very minute cocci, ovoid in shape, and in rapid molecular

motion. Many are seen in pairs, and a few in temporary short chains of four, the latter more especially if they have been growing on alkaline agar. The chains are never seen in dried cover-glass preparations. They stain very readily with all the aniline dyes, but lose their colour very rapidly if treated with alcohol or any of the usual decolourising agents.

The slow growth, peculiar appearance on agar, the small size that the individual colonies attain, the minute coccus, and the readiness with which the micro-organism decolourises, serve to differentiate it from others.

Inoculation Experiments.

(*a*) *By Bruce.*—(1) A male Bonnet monkey was inoculated in the left forearm with growth obtained from the human spleen, by means of a perfectly clean Pravaz syringe, the growth having been growing on artificial media in a pure state for a month previously. No changes appeared at the seat of inoculation, but on the day after his temperature began to rise,[1] and reaching 106° to 107° F., he died on the twenty-second day from inoculation. On post-mortem examination the lungs showed no signs of tuberculosis ; the liver and spleen were enormously enlarged, and the intestines were free from ulceration.

Six tubes of agar were inoculated from the spleen, and two from the liver with full precautions. In all those from the spleen, and in one of those from the liver, the *Micrococcus miletensis* appeared after 168 hours. In the remaining tube no growth appeared.

(2) Another male monkey (Bonnet species) was similarly inoculated. The temperature rose rapidly, and he died on the thirteenth day. Growths were obtained from the internal organs, making their appearance after four days.

Bruce further found that rabbits, guinea-pigs, and mice, gave negative results.

[1] See 'Practitioner,' xl, 1888, p. 241.

(*b*) *By me.*—(3) A small male monkey (Bonnet species) was kept under observation for two months, during which time his appetite was good and his temperature stationary (about 99° F.). He was inoculated in November, 1891, by the injection of a colony of *Micrococcus miletensis* obtained from the spleen of the the first of the writer's cases, which had been growing in a pure state in the incubator for three weeks. The site of inoculation (the muscles of the forearm) had been previously shaved, cleaned with soap and water, and washed with a solution of perchloride of mercury. The syringe and all other apparatus had been carefully sterilised; the growth being mixed with 1 c.c. of sterilised bouillon, which had been under observation for one month previously.

Forty-eight hours afterwards the temperature began to rise, and daily increasing with a remittent curve, reached 106° F. on the fifteenth day, when he was killed, 362 hours after inoculation. For the first ten days the monkey was lively and continued to eat his food, but after that he lay about and refused it. See further, Chart XIII.

Five minutes after death the lungs were found to contain sero-purulent exudation in the bronchial tubes; the liver was congested; the spleen congested and very large in proportion to its body weight; there was slight congestion near the ileo-cæcal valve, but Peyer's patches were quite normal, and no other pathological condition was present. Seven tubes of agar were inoculated from the spleen in the usual manner; two tubes were inoculated from blood collected with an aseptic syringe from the centre of the unopened heart. These were placed in the incubator at 37° C., and of the seven splenic cultures five showed colonies of the *Micrococcus miletensis* in 168 hours, two showed contamination with *Micrococcus albicans* the next day; while of the blood-cultures, one showed the tppical growth after 168 hours, the other proved sterile. These growths were identical with those obtained from human spleens, macroscopically and microscopically, were

cultivated in bouillon, were carried through six generations of pure cultures on agar without change, and were identified by Bruce at Netley.

(4) A small African monkey was inoculated in the muscles of the forearm with growth from the cardiac blood of the last monkey, the same precautions being taken. The temperature began to rise the following day, though it had remained steady for a month previous to inoculation, and for eleven weeks he suffered from remittent pyrexia (see Chart XIV). He finally made a complete recovery.

(5) A female monkey (Bonnet species), under observation and in good health for three months previously, was similarly inoculated in the muscles of the right thigh with virus obtained from the spleen of the writer's first monkey, which had also been for some time under observation in the incubator to insure its purity. This monkey developed pyrexia consisting of intermittent waves of a remittent type, lasting over ninety-four days. She ultimately made a good recovery, and lived in good health for three years until killed by a dog. During the attack she lost weight, and like the last case seemed to suffer from pain or rheumatism of the extremities at irregular intervals, with irregularity of the bowels (see Chart XV). These monkeys' temperatures were taken three times a day by the same two persons throughout, a large amount of petting, time, and thermometers being expended. In no case did any changes occur at the seat of inoculation.

(6) A very savage and impatient monkey was also inoculated with human virus, and his temperature was taken at various intervals. No changes occurred at the seat of inoculation; he suffered severely from pyrexia, and ultimately recovered, but little more can be said.

Thus it may be said that when pure growths of this micro-organism are introduced into the tissues of healthy monkeys a pyrexial condition closely analogous to the Mediterranean fever of man is set up, and that after

death a similar micro-organism can be isolated from their tissues in a pure state which is capable of producing a similar disease in other healthy monkeys.

More experiments are needed, but unfortunately neither the time nor the money necessary are available. The above experiments, however, fulfil the requirements of Koch's postulates entirely.

(*b*) *Age and sex.*—All ages are liable to this fever. Among soldiers and their families the ages have usually varied between five and thirty years. Infants under two have not often suffered. In the old long-service days at least half the cases were over thirty. Among the inhabitants of Italy and Sicily Guiffré gives between fifteen and forty years as the most susceptible ages, and states that children under six and adults over fifty are relatively exempt. This would appear to agree with the cases among the native population of Malta. Though no rule is absolute the average age of enteric cases in Malta among soldiers is lower than that of Mediterranean fever.

In a given number of families the women are attacked oftener than the men, the reason appearing to depend rather on age and occupation than on sex.

(*c*) *Season and climatology.*—In Malta the admission rate is lowest during the first quarter of the year, rises rapidly in May to a maximum in July, August, and September, after which it gradually falls until November and December, which two months are somewhat higher than those of the first quarter (abstract of 1339 cases). The same is the case in Gibraltar, Rome, Palermo, and Cyprus.

In Malta and Gibraltar its prevalence has been found to be in an exact inverse ratio to the amount and continuance of the rainfall from month to month. While the surface of the ground is kept constantly wet by rainfall the admission rate keeps down, but the cessation of rain in warm weather is at once followed by an increased activity of the poison, and a sudden excess in the attack

rate. Since 1859 the greatest prevalence has been during the driest summers, while the attack rate has become of increasing importance when the average monthly temperature has exceeded 60° F., and the rainfall descended below two inches a month (see Chart XI). The attack and mortality rates have shown a tendency to form quantitative waves of about seven years' duration.

(*d*) *Length of residence.*—This does not appear to confer immunity, for while in Malta, Gibraltar, and elsewhere enteric fever specially favours new-comers, Mediterranean fever attacks those who have been in residence for even six or more years. When a regiment is placed in an unhealthy barrack, if the susceptible element is very great, it suffers accordingly, nor does previous Mediterranean service confer an immunity.

(*e*) *Mode of prevalence.*—The disease is not propagated by contagion from man to man, nor does it appear to have any causal connection with milk or food supply. The distribution of the main water supplies does not in any way account for its localised distribution, and whatever effect polluted tank water may have on the native population, many hundreds of soldiers and Maltese are attacked, who have no access to such water. The season of prevalence and the distribution of cases of water-borne enteric fever in Malta are quite different from those of Mediterranean fever, and when both fevers occur together a dual cause is found to be at work.

In the past, Marston, Boileau, Chartres, Donaldson, Oswald Wood, Notter, De Chaumont, Duncan and Moffet, have ascribed this fever to insanitation, and more particularly to defective house drainage and fæcal pollution of the soil, the latter giving an accurate illustration of an outbreak at Gibraltar from such causes. In Italy opinions are divided, but those who disbelieve in a fæcal origin give no other explanation.

In my experience nearly all cases are localised, even when in epidemic form, to certain rooms in certain buildings, which rooms yearly produce their fever cases

until some insanitation is rectified; the most constant factors present being fæcal contamination of soil (and air) by persons suffering from the fever, together with warmth and moisture; the factors necessary for its diffusion being the subsequent drying of the soil, the presence of air currents, and the close proximity of the human subject (a question of from two to twenty feet perhaps). Numerous outbreaks, confined to one household, have occurred simultaneously with the flooding of the floors with sewage from a choked and burst house drain, or overflowing cesspit. Many sporadic cases can be traced to escape of sewer gas from neighbouring drain ventilators, which, though above one roof, may be on a level with, and only a few feet off the windows of neighbouring houses, or to direct communication between rooms and the main drains by means of untrapped sinks or cupboard latrines; the virus being often concentrated in such cases by insufficient room ventilation. Occupants of healthy houses are exempt, but come under its influence on moving into infective areas. It is most difficult in so short a space to deal with this important and necessarily much-debated subject; especially as the evidence is, like that of all early epidemiological investigations, of a somewhat circumstantial nature; but, as Bishop Butler has said, "probability is the very guide of life." Out of a number of instances, the following will serve to illustrate the above points.

(1) In a new and well-constructed flat in Valetta the first occupant was invalided home with this fever. The next also suffered from the same fever, together with his wife and child; the child died, and the man and woman were invalided home. The next occupant was a man with a wife and child. The sister suffered severely and the wife [illegible]. An inspection of the quarters [illegible] latrine on the verandah [illegible] drain ventilator running in [illegible] with the same [illegible]

was ten feet distant from the bed, entered the room, and was most offensive. People of the same rank in life occupying other portions of the block were unaffected. The water-supply was excellent and common to the whole of Valetta; the milk was obtained from goats milked at the door. The latrine was rectified and the ventilator placed outside the building, with excellent results.

(2) A regiment was quartered on a small island in one of the harbours of Valetta from January 2nd, 1892, until October 10th of the same year. During this period it suffered severely from "simple continued fever," 197 cases being admitted from a total strength of 760 men. The regiment had suffered severely from this fever elsewhere during its first year of service in Malta, had been seasoned by three years' residence, and was composed of men whose ages were not below the average of the station, yet their fever rate for 1892 far exceeded that of any other regiment. Of these men 480 were quartered in wooden huts, and the remaining 280 in an old fort close by, which had been built by the Knights in 1775. A careful analysis of the fever admissions divides them into two classes:

(*a*) Cases of true Mediterranean fever.

(*b*) Relapses or slight cases of the same fever (?), cases of simple ardent fever (febricula), and other obscure but slight febrile ailments without localised symptoms. The latter class were in hospital but a short time, and there is little to note except that the numbers were greater than from any other barracks in the island, and that in proportion to strength the admissions from the fort were double those from the huts.

The admission rate per mille for true Mediterranean fever in Valetta district (strength 3511, including the island in question) in 1892 was 52·2, while those of the [illegible]s and fort in question were 46·3 and 178·6 respectively, [illegible]ing that some local cause of fever must exist within [illegible]rt. The men in the fort and huts belonging to the

same regiment were of the same age and class, and under identical conditions as regards food and water supplies, the latter being common to a very large area of unaffected population, civil and military. In the fort thirty-eight men, two women, and five children were affected; and of the men one died (see Chart IX, Case 3), six were invalided, of whom one was finally discharged from the service, fourteen were sent to the sanitarium, and seventeen returned to duty straight from hospital, but were in many cases readmitted with relapses after leaving Valetta in October. The average stay in hospital was 109 days each for the men. The majority of the rooms in question were dark, close, and damp, and were never intended for barrack rooms. Round the back of the rooms ran large channels cut in the extremely porous rock, and passing on each side of the fort down to the sea. From 1870 these channels were used as sewers until the substitution in 1885 of the present dry-earth system. Mediterranean fever occurred in the fort as an epidemic in 1870 and 1872, but was not excessive from 1885 to 1891. During the latter period a quantity of sewage remained boxed up, with the result that the stone became soaked with sewage, even through the walls and flooring to the adjacent rooms. Analysis of portions of the walls of the channels and rooms showed a larger percentage of chemical constituents of sewage, much organic matter, and a very large number of putrid and non-pathogenic organisms, when compared with similar but unpolluted stone. Again, though unpolluted stone was highly alkaline, the stone of these walls was neutral or faintly alkaline, forming therefore a suitable nidus theoretically for the micrococcus of this fever. Between September and December, 1891, a thorough overhauling of the drains took place, these channels being cleaned and converted into surface-water drains, gratings being placed at intervals almost on a level with, adjacent, or even opposite the windows of the barrack rooms. Owing to their situation and construc-

tion there was a varying yet constant current of air from the sea travelling up these channels, and passing out of the gratings into the fort, the porous walls at the same time being wet or dry according to the state of the atmosphere and the amount of rain. There was ample opportunity for miasmata to pass from the rock channels into the barrack rooms, and it is a significant fact that these forty-five cases slept in beds grouped in close and definite relation to the rock channels and sewage-soaked walls, and in direct proportion to the amount of varying dampness present. The first case was admitted four days after the cessation of the heavy rain. There was no other apparent cause for the outbreak, and obvious sanitary measures have resulted in a cessation of this fever prevalence.

(3) In a large modern and well-built hospital, standing on one of the best sites in Malta, a number of cases of this fever (20—40) have for many years past been treated in wards on the top floor, without the disease spreading to others in the same wards. The hospital had always been considered a very healthy one until recently, when a few cases of this fever began to occur among venereal patients on the middle floor, and the patients and sick attendants occupying the ground-floor suffered severely from this fever. In the kitchen behind the hospital two cooks died of its effects, and patients suffering from slight ailments, such as sprains, &c., and who occupied tents immediately behind the building, also developed the fever. Many of the sufferers had been in hospital over a month, while others had resided in the building for many months previously. The water-supply was good and common to a large district, the milk-supply was above suspicion. On investigation it was found that the hospital drain-pipe, which ran along the back of the hospital between the main building and the ground-floor occupied by the kitchen and tents, had been blocked for some time beyond the main building, and that the fæcal

sewage from the fever and other wards had forced the joints of the pipe and leaked in all directions under the hospital foundations, and through the ground on which the kitchen and tents were situated.

At the same time in three officers' quarters in the same grounds, but well detached and for many years considered to be among the healthiest in Malta, six cases of this fever occurred in one summer. In this case the main and house drains were found blocked, and the backflow of sewage caused offensive odours of sewer gas to enter the back rooms from adjacent drain ventilators.

Though the details are not exactly laid down in this last instance, I have preferred it to other smaller instances with exact details on account of the large number of cases occurring in an English-built hospital, which had for a number of years been healthy.

Numerous sporadic cases occur in the old barracks built by the Knights during the last century, the causes of which are not always apparent. When we remember the state of filth in which these buildings must have been in those days, the soft porous stone with which they are paved, and of which their foundations are made, and, moreover, the way in which the whole foundations of the towns have during the past been riddled with leaking sewer conduits and cesspits, with the denseness of the population in Maltese and other towns, we can only wonder that the disease is not more prevalent. As these sporadic cases occur over and over again in the same rooms, doubtless further research will extend our knowledge.

Lastly, I wish to draw attention to a theory that the soldiers and sailors catch this fever while bathing in fæcally polluted harbour water. This could only account for a portion of the cases [illegible] is at present but a matter of opinion, unsupported [illegible] data, and needing further inve[illegible]

(*f*) *Im*[illegible] [illegible] ugh the medical

histories of many thousands of soldiers have been examined —these histories being accurately kept official documents in the case of every enlisted soldier—no record but one has been found indicating two attacks of this fever in the same individual. Though this may be owing to the short period that men are now stationed within the infective area, yet it has not prevented men from suffering from both enteric and Mediterranean fever on separate occasions. Guiffré does not believe that it confers immunity. From its behaviour to native Maltese it is probable that a temporary but not an absolute immunity is conferred, comparable to that met with in attacks of enteric fever.

Diagnosis.

Enteric fever is the most important disease, from a diagnostic point of view, that this fever can be mistaken for. The mistake is most likely to be made in severe cases with a tendency to pass into the so-called "typhoid state." Mediterranean fever is specially marked by its uncertain duration, irregular course, and the tendency in many cases to approach or reach normal during the first week, without apparent reason such as hæmorrhage, &c. (see Chart X); by the absence of hæmorrhages, rash, and iliac tenderness; by the rarity of diarrhœa and meteorism, by the physiognomy, moist tongue, smell, and an indescribable something about the fevers felt by the experienced; and lastly by the peculiar diaphoresis, neuralgic and rheumatic symptoms, and an absence of a rose-red reaction with Ehrlich's urine test.

From *paludism* it is distinguished by its non-paroxysmal nature, its resistance to quinine, and by the entire absence of the hæmatophyllum of Laveran.

From *phthisis, liver abscess,* and other suppurating diseases it is distinguished by the want of any local lesion to account for an apparently hectic temperature.

Prognosis.

This is good as regards danger to life and ultimate recovery, the case mortality being under 2 per cent.; as regards the length of time on the sick list, however, it is very unsatisfactory, the average time spent on the sick list being about ninety days. A previous history of cardiac or pulmonary disease, the presence of organic cardiac or renal disease, of excessive pyrexia, intermittent heart's action, pericardial effusion, diarrhœa from involvement of the large gut, broncho-pneumonia, tendency to dry brown tongue, and bedsores are all grave symptoms calling for constant attention and a guarded prognosis.

Pathological anatomy:

Based on reports of sixty post-mortem examinations on soldiers (duration of disease 4 to 156 days), at thirteen of which I was present. The general congestive appearances in cases which proved fatal during the first thirty days will, when necessary, be kept distinct from the generally more localised lesions in later cases.

Brain.—In thirteen cases in which the cranium was examined, the meninges and choroid plexus were congested (most marked in early cases), and in some there was effusion into the ventricles.

Heart.—The muscular walls are described as pale and flabby. In two cases (duration seventeen and sixty-two days) there was thickening of the mitral valves with pericardial effusion, the latter being the actual cause of death. In three short and three long cases mitral vegetations were present, and in one of the latter (duration 156 days) there were also aortic vegetations, a small aneurismal dilatation at the root of the aorta, and old pericardial adhesions.

Lungs. [illegible] basal pneumonic congestion [illegible] lobes, with serous

or sero-purulent exudation, were present. In 80 per cent. of early cases this had gone on to lobular consolidation, generally more marked on the right side. In 25 per cent. of cases there were pleuritic adhesions on one or both sides, in early cases apparently of former origin, but in later cases often undergoing vascular organisation. The bronchial glands were enlarged in proportion to the lung mischief present.

The *alimentary canal* is subject to patches of congestion, which, however, are not specially characteristic of this fever. These occurred in the stomach in 18 per cent., in the duodenum in 17 per cent. of short cases; and in the small intestines in 68 per cent. of all cases; while in the colon the condition occurred in 27 per cent. of all cases, of which thirteen had that peculiar swollen, inflamed, and œdematous condition of the mucous membrane which characterises certain severe cases of this fever. The congestion occurs in patches, following the arborescent course of the vessels, is not confined to nor constant in any one situation, and has no relation to Peyer's patches, which latter are unaffected. In early cases the mucous membrane is swollen and softened, but in late cases the intestines become extremely attenuated, the muscular and mucous coats having shrunk.

The *mesenteric glands* are enlarged in proportion to the intestinal congestion, being of normal size in most of the late cases.

Liver.—In half the cases this is congested and slightly enlarged, while in very late cases it is often nutmeg.

Spleen.—The spleen is always enlarged and congested. In early cases its weight averages 18 to 19 ounces, of a dark reddish-black colour, and in some 70 per cent. of such cases it is soft and friable like blood-clot, and in about 18 per cent. it is absolutely diffluent. In late cases the average weight is about 15 ounces, and it is of firmer texture.

Kidneys.—These are congested in early cases and often slightly enlarged; while in two long cases a condition of

large white kidney was present. The capsules are usually somewhat more adherent in long cases.

Microscopical appearances.—The microscopical appearances are scarcely characteristic, but are more or less common to other acute fevers. In acute cases, according to Bruce, "the Malpighian bodies of the spleen are enlarged from an apparent increase in the number of the round lymphoid cells; the endothelial cells of the marginal sinuses are proliferating and swollen; a condition of intense congestion is seen in the section, the sinuses being enormously distended with blood; there is a marked exudation of small round cells along the lines of most of the venules. The liver is congested, the cells in a condition of cloudy swelling, and there is an infiltration of small round cells in the interlobular fissures. The kidney is congested and in a state of glomerular nephritis. The mesenteric glands when enlarged show proliferation of the cellular elements of the lymphoid tissue. The only change in the Peyer's patches is a slight proliferation of the cellular elements of the mucous and submucous layers."

Dr. R. T. Hewlett, of the British Institute of Preventive Medicine, has kindly examined the tissues of Case 12, in which death occurred on the 111th day of the pyrexia. He reports as follows:

"*Heart* (mitral valve), a small vegetation is present, which has become nearly fully organised into fibrous tissue.

"*Lung,* many of the air-vesicles contain large catarrhal cells and shreds of fibrin.

"*Liver,* neither fatty nor fibroid change; some slight amount of cloudy or granular degeneration of the liver cells.

"*Spleen,* nothing abnormal detected. *Mesenteric glands,* ditto.

"*Peyer's patch,* a slight increase in the lymphoid cells, no ulceration.

"*Kidney,* some tubular nephritis, evinced by cloudy swelling of the protoplasm and loss of nuclei of the epithelial cells of the tubules. In some places there is

some infiltration of round cells into the intertubular tissue. The glomeruli are normal.

"These changes are such as would be met with in the lungs, liver, and kidneys of any case of severe and prolonged fever."

Prophylaxis.

The institution of sound water drainage for all new houses when first built, constant and efficient flushing of both main and house drains, the raising of all drain ventilators well above and away from all windows, the abolition of all leaking cesspits in porous soils in thickly populated districts, the paving of cellars, yards, and in some cases of the streets, and the proper trapping and disconnection of all sinks, &c., the avoidance of polluted ground for camping purposes, and the prevention of pollution to the ground round camps and buildings, and due attention to all latrines and urinals, proper ventilation, and avoidance of damp and overcrowding. I here assume that the virus is of an aërial and fæcal nature—a conclusion I adopt from the nature of the virus, the season of its prevalence, and the distribution of cases. Finally, avoid Mediterranean towns between the months of June and October, and send all women and children away when possible during those months, and always remember that the Mediterranean, except in the hot summer months, is subject to sudden changes of temperature and that the air is very moist, necessitating the possession of the warmest of clothes, an extra cloak at sundown, and teaching the desirability of always sleeping in flannel, and wearing it next the skin.

Treatment.—There is no specific drug at present known which will cut short an attack of this fever. The treatment consists of placing the patient under the best circumstances for Nature to effect her own cure. Although many cases do well with careful nursing and dietary alone, it is a great mistake to think that that is all that

is necessary, for we each find that as our experience increases our deaths decrease, our cases become milder and shorter, and complications are less frequent.

The patient should first be removed from any insanitary surroundings; he should be confined to bed entirely, and placed on fluid diet, as would be done in the case of enteric fever, bearing in mind that beef tea is incompatible with the presence of diarrhœa. Lemonade made from fresh lemons, grapes, or lime juice should be given to prevent any tendency to scorbutic symptoms, while soda water and fluids should not be restricted. If dyspeptic symptoms, nausea, or vomiting be present lime water should be added to the milk, and bismuth given internally; while if this condition becomes serious beef-juice, peptonised food, and champagne are very useful. There is a great tendency to overfeed patients with high temperatures. Patients should have abundance of fresh air, but be screened from direct draughts, and on account of the tendency to lung symptoms care should be taken to lay the dust with tea leaves before sweeping, especially in the case of the soft stone floors of Malta. All patients should wear flannel or flannelette sleeping suits, which in the case of children should be in the form of "combinations." As these will need frequent changing during the night a good supply should be available. Sheets should be of cotton.

Should the diagnosis be sure, the bowels should at once be opened. For this nothing works better than a good dose of calomel, combined with Pulvis Jalapæ, which frequently alone is sufficient to reduce the temperature in a constipated case. The bowels must further be kept open every oth[illegible] at least, with occasional doses of calomel, cascara, [illegible] Glycerrhizæ Co., or by enemata where [illegible] exists. As diarrhœa is usual[illegible] the large (or less often of the [illegible] tends rather to prevent than [illegible] appears to prevent the n[illegible] later

stages when persevered with at first. If diarrhœa be present, the greatest care must be taken to prevent it becoming serious. The diet must be attended to, and in the case of the large bowel, enemata of starch and opium must be given frequently. These enemata should be made with sufficient boiled starch to produce a consistency like cream, be given warm, and retained as long as possible. Rectal irrigation with a solution of boracic acid has been advocated. Diarrhœa of the small bowel is often due to unsuitable food or medicine, when otherwise it may generally be checked by a lead and opium pill, or by a mixture of opium, aromatic chalk powder, and astringents. The teeth, gums, and tongue must be attended to, and in summer a net is useful to keep off the flies which swarm round severe cases.

The skin should be sponged daily with tepid water and acetic acid, while a warm dose of diaphoretic mixture and brandy at night is often beneficial when the skin in severe cases remains dry. Sudamina and prickly heat should be bathed (but not rubbed) three times a day with a solution of boracic acid, while the extreme irritation of the latter may often be allayed by a coating of mild soap, left to dry on the surface until the next application of the boracic solution. Precautions should be taken against bedsores, especially towards the end of an attack, and when boils are present. The pulse must be carefully watched, its condition being taken as an index of the amount of stimulants necessary. When intermittent or weak in action, small doses of strychnia and digitalis with the exhibition of good champagne have worked well. In two cases of effusion into the pericardium without other complication, aspiration was not tried, and both proved fatal. Sleeplessness and nervous irritability are treated by a full dose of morphia with bromide of [illegible] at night, or by Dover's powder when diaphoresis [illegible] affection of individual nerves by the [illegible] flannel, and cotton wool, with perhaps [illegible] locally. Chronic sciatica during

convalescence by constant flannel next the skin, warm ironing, gentle massage and rubbing with counter-irritant liniments. Painful and acutely swollen joints are soon relieved by continuous hot fomentation, followed by wrapping in cotton wool and flannel; acute orchitis by warm hip-baths, followed by support and belladonna. The painful hyperæsthetic condition of the feet yields best when soaked in cold water or wrapped in cold water bandages, any application of heat or warmth being intolerable. Lung complications should be at once treated with sal volatile, alcohol, and other stimulating expectorants, and when severe by jacket poultices, which followed by a cotton-wool jacket are most efficacious.

The bladder must be watched in severe cases lest retention be missed, while any irritability is generally removed by the administration of acetate of potash, soda water, and saline diuretics.

One of the greatest advances, however, in the treatment of this fever consists in the moderate regulation of the pyrexia by the application of tepid, cold, or iced water externally. By this means the commonest cause of death (hyperpyrexia) is avoided; the exhausting effect of high temperature on the heart, respiration, and higher nerve-centres abated, and death from these causes postponed or averted; chest and other complications become less common, while severe cases are converted into mild ones. We find that this form of treatment, while acting strongly on a special symptom, at the same time by stimulating the circulation and respiration, the metabolic and eliminatory processes, improves oxygenation, and aids in the elimination of toxic substances from the blood. At the same time, by giving rest to the heart, it restores equilibrium to the internal organs, increases the resistance and fighting power of the body, and so without injuring the patient has an almost specific action in this fever, beyond that met with in any other form of pyrexia.

To produce a satisfactory result, this treatment must be begun early, and before a fatal result is anticipated, or

the patient may be found to have already lost the strength necessary for successfully combating with the disease. The form of application which I have found most satisfactory has consisted of keeping the temperature systematically below 103° F. by means of cold water sponging, the application of iced water or ice packing according to circumstances. A great deal may be done in all cases by tap-water sponging, but ice packing should always be supervised by an experienced individual, the temperature being taken frequently and the pulse watched. Bad results have not followed this treatment when reasonably applied. The great secret of success lies in the avoidance of too great reductions, the temperature being reduced 2°—3° F. to a safe range, and no further. The administration of stimulants before and the avoidance of chills after treatment should be borne in mind, and also that no two patients respond with the same rapidity to this treatment. The immediate relief of headache, the refreshing sleep which follows such reduction, are marked; while the return to consciousness from hyperpyrexial coma is too well known in other diseases to need description.

The cleaning of the tongue and shortening in duration of the daily pyrexia are indications that the time is approaching for the cautious increase of diet, and that the patient may be soon allowed up for an increasing amount each day; but in most cases, and always when acute, this is better postponed until the temperature has been normal for at least ten days. The mucous membranes take a considerable time to recover their full function. During convalescence Stout, Blaud's pills, and the Citrate of iron and quinine are useful. Change to a warm, dry inland place in England is very beneficial during the enervating heat of the summer, but the trials of a sea voyage or overland journey should not be encountered until the acute stages are over, nor until an enteric diagnosis has been excluded. During the autumn and winter months patients are best away from the rigor of the English climate, and most cases recover well if

kept in Malta or sent to the dry atmosphere of Cairo or to the hill towns of Italy or Sicily at that time of year. It is not justifiable to send acute cases away from the attentions and comforts of home or hospital, to the fatigues of travelling, the doubtful comforts of foreign hotels, and away from skilled and friendly advice, for the sake of an over-estimated treatment by change of air, and often to places where the same fever is known to exist. The beneficial effect of fresh air and change of environment during convalescence is, however, not to be denied.

Quinine in acute stages acts as an acute stomachic irritant and depressant, and does more harm than good. In very late stages when the appetite has returned, and the patient, though up and about, is still subject to

slight nocturnal
three times a day

steadying the temperature. Arsenic is injurious in acute stages, aconite dangerous from its effect upon the heart. Antipyrin at the beginning of an attack reduces temperature, relieves headache, and promotes perspiration, but is inferior in action to sponging, and in severe cases has a most dangerous effect upon the heart. Germicides, such as carbolic acid, boracic acid, and mercury, have been tried without proving of specific value.

In conclusion I may point out the importance to the State of the study of this fever. Its prolonged duration and high invaliding rate cause a very large amount of non-effectiveness among the 25,000 soldiers and sailors composing the Mediterranean garrison. In the Malta garrison in 1891, the loss to the State on account of this fever alone was equal to the loss of the services and cost of a whole regiment 1000 strong in hospital for twenty-five days. The first step needed is to give this fever a place in the Official Nomenclature of Diseases.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. viii, p. 176.)

EXPLANATION OF CHARTS.

CHART I.—Case of undulatory Mediterranean fever, admitted during the primary wave, with constipation (P. = aperient) and well-marked waves. Pyrexial duration, eighty-six days.

CHART II.—Similar case, with more regular waves, admitted to hospital in December, 1891, after nineteen days' treatment in quarters. Suffered from obstinate constipation (E. = enema), anorexia, anæmia, and extreme debility, but was never dangerously ill. Invalided to England on the 114th day of the pyrexia, where he suffered from occasional rises of temperature and severe rheumatic symptoms. Returned to duty in Malta in January, 1892, and has remained perfectly well since but for a slight attack of febricula in the summer of 1892, lasting three days.

CHART III.—Mediterranean fever with prolonged wave, associated with symptoms of cerebro-spinal irritation (P. = aperient).

CHART IV.—Chart of Case No. 9, "Bacteriology."

CHART V.—Illustrates a final wave of excessive symptoms at the end of a long case.

CHART VI.—Case of intermittent Mediterranean fever with constipation (P. = aperient), night sweats, malaise in the afternoon and evening, but no other complicating symptoms, ending in complete recovery. Treated on separate occasions with quinine (large doses once a day and also with small doses three times a day), arsenic, salicylates of quinine and soda, and antipyrin, without apparent effect.

CHART VI (a).—Complete diurnal curve, same case, days 43—51, with usually taken morning and evening curve in dotted lines.

CHART VII.—Mixed case commencing with the undulatory and ending with the intermittent type.

CHART VIII.—Case 2 in "Bacteriology."

CHART IX.—Case 3 in "Bacteriology."

CHART X.—Early sudden pyrexial drops in cases of Mediterranean and enteric fevers contrasted.

CHART XI.—Admission rate and rainfall contrasted.

CHART XII.—A common type of case with the waves less clearly marked.

CHART XIII.—Monkey experiment No. 3 (Hughes).

CHARTS XIV AND XV.—Pyrexial charts, monkey experiments 4 and 5 (Hughes), inoculated from last.

OBSERVATIONS AND EXPERIMENTS

ON

INTESTINAL AND GASTRO-INTESTINAL ANASTOMOSIS

(FROM THE BROWN INSTITUTION)

BY

WALTER EDMUNDS, M.C.,

AND

CHARLES A. BALLANCE, M.S.

Received March 20th—Read May 12th, 1896.

SYLLABUS.

I. Intestinal anastomosis.
  A. Side to side.
  B. End to end.
  C. End to side
  Choice of operation for:
  A. Artificial anus.
  B. Gangrenous hernia.
  C. Malignant disease of bowel.
  D. Intussusception and volvulus.
II. Gastro-enterostomy.
III. Physiological observations.
  1. Shortening of blind ends in lateral anastomosis.
  2. Short circuiting experiments.
  3. Changes in the mucous membrane.
  4. Implantation experiments.
  5. Reversal experiments.
IV. List of experiments.
V. References.

THE object of the experiments here given was to determine the best method of uniting either two parts of intestine together, or the stomach to intestine.

According to Senn, Maisonneuve was the first to attempt to make a fistulous opening between two portions of bowel,—that is to say, an intestinal anastomosis between a portion of bowel above, and another below the seat of obstruction. Unfortunately both Maisonneuve's cases were unsuccessful.

From time to time the subject was touched on by surgeons, but it is to Senn's numerous experiments, and his energetic advocacy (in 1887) of these operations in competent hands, that the recent progress in this department is chiefly due.

I.—Intestinal Anastomosis.

There are three main methods of uniting two pieces of intestine, namely,

(A) **Side to side** (by lateral openings),

(B) **End to end,** or

(C) **End to side,** *i. e.* by the implantation vertically of one piece of intestine into another—this last being useful in the treatment of cases where the obstruction cannot be removed.

All our experiments were made on dogs. It will, no doubt, be objected that the dogs were in good health, and not suffering from the effects of intestinal obstruction, and that the intestines to be united were healthy, and not affected either with distension from obstruction or with inflammation.

These objections are no doubt to some degree valid, but with respect to both obstruction and inflammation it must be remembered that it is not imperative in most cases in man to perform a primary anastomosis.

It will further be objected that the dog's intestine is so different from that of man as to vitiate any cross-inferences from experiments. Figs. 1 and 2 show transverse sections of human and dog's small intestine. They are the same magnification, and it will be noticed that the dog's intestine is more muscular, but that the submucous coat is about the same in both. This submucous

coat is very important on account of its strength, and it is in it that Halsted insists that the sutures should get their hold.

The submucous coat is important in surgery in another way, for it is of it that catgut for surgical purposes should almost exclusively consist. Lister, in his account of the

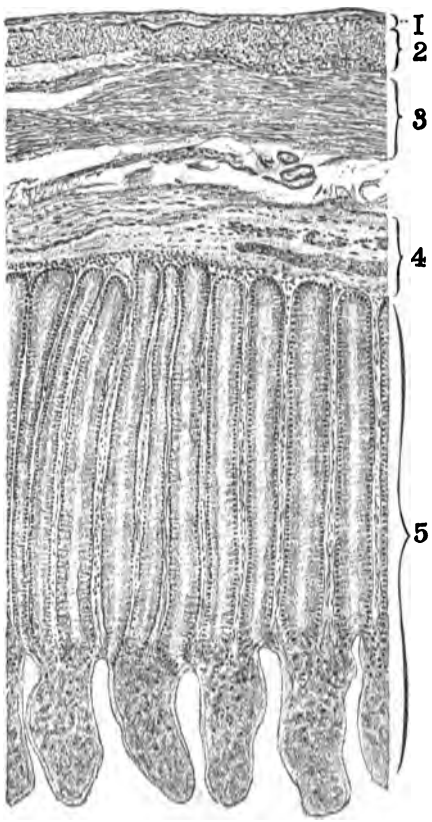


FIG. 1.—Transverse section of human small intestine. (×100.)

1 = Peritoneum.
2 = Longitudinal muscular coat.
3 = Circular muscular coat.
4 = Submucous coat.
5 = Mucous membrane.

Between 3 and 4 is some loose tissue, allowing of some gliding movement.

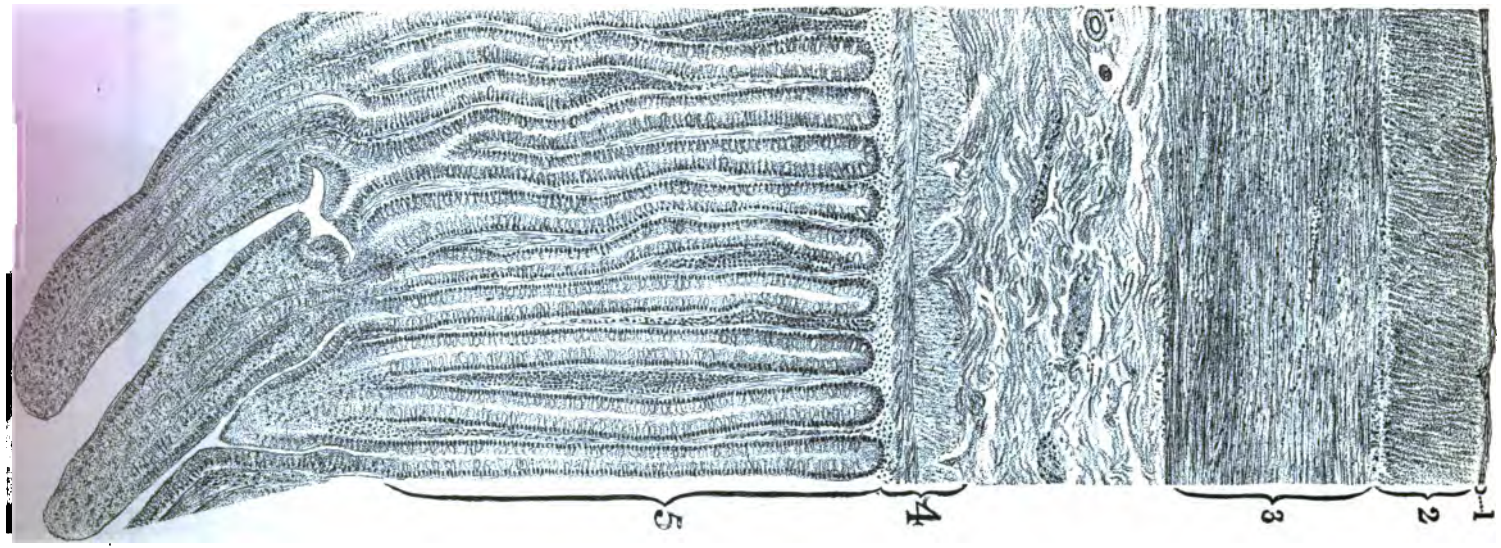


FIG. 2.—Transverse section of small intestine of dog. (× 100.)

1 = Peritoneum.
2 = Longitudinal muscular coat.
3 = Transverse muscular coat.
4 = Submucous coat.
5 = Mucous membrane with villi.

Between 3 and 4 is some loose tissue, allowing of some gliding movement.

preparation of catgut, relates how the sheep's intestine is spread out, the mucous membrane scraped off, and the muscular coat also removed, and thus the submucous coat alone is left.[1] All catgut, however, is not prepared

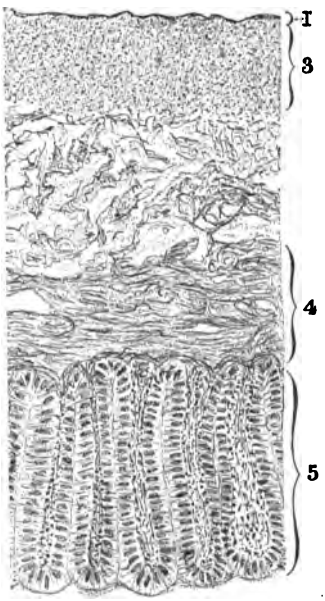


FIG. 3.—Transverse sections of the human large intestine : × 100. It will be noticed that the submucous tissue is particularly thick. As the section is not made through a band there are no longitudinal fibres shown.

1 = Peritoneum.
3 = Circular muscular fibres.
4 = Submucous coat.
5 = Mucous membrane.

[1] The method of preparation of catgut is thus described by Lister:—"Catgut, as you are doubtless all aware, is prepared from the small intestine of the sheep. The gut is treated in what seems an exceedingly rude manner for so delicate a structure. It is scraped with some blunt instruments, such as the back of the knife, over a board; and by this means, as the people express it, the dirt is scraped out. That which these people call the dirt is the exquisite and complicated structure of the intestinal mucous membrane. But while the mucous membrane is scraped out from within, there is also

in this way, for on microscopic examination of some of it the mucous membrane can be seen.[1]

Fig. 3 shows a transverse section of human large intestine. The section is not carried through a band of longitudinal fibres; the submucous coat is decidedly thicker than in the small intestine.

The following table gives the thicknesses of the various coats:

Measurements in hundredths of a millimetre. The specimens had previously been hardened for microscopic purposes:

	Small intestine, dog.	Small intestine, human.	Large intestine, human.
Peritoneum . . .	1 ...	1 ...	1
Longitudinal muscular coat	12 ...	5 ...	—
Transverse muscular coat .	27 ...	10 ...	12
Loose tissue . . .	(25) ...	(5) ...	(20)
Submucous tissue . .	10 ...	10 ...	15
Mucous without villi .	70 ...	50 ...	32

It must, however, be admitted that as the subject of surgery is man, man is, scientifically speaking, the proper animal to experiment on—and this is what practi-

scraped off from without the circular coat of muscular fibres. The result comes to be that the intestine is converted into a comparatively unsubstantial material consisting of two parts or bands, one more slender than the other. When the mesentery is stripped off by the butcher the peritoneal covering of the gut shrinks into a narrow strip, and this, with some longitudinal fibres, constitutes the more slender of the two parts to which the intestine is reduced by this process of scraping. The other part is the essential material from which the catgut is prepared, and this is neither more nor less than the submucous cellular coat of the intestine. When I first visited a catgut manufactory I was astonished to find that after this scraping process the intestine could be blown up still as a continuous tube, as you see can be done with this specimen, which has been treated in the manner I have described. This exquisitely delicate structure is a beautiful anatomical preparation of the submucous cellular tissue, though made in so rude a fashion. This coat of the intestine, which in the sheep has this extraordinary toughness, is the material out of which the catgut is prepared."—'Lancet,' Feb. 5th, 1881.

[1] See Ballance and Edmunds, 'Ligation in Continuity,' 1891, p. 249, fig. 105.

cally we should be left to if there were no experiments on animals; but as by the latter more strictly scientific methods can be followed, the progress of our knowledge

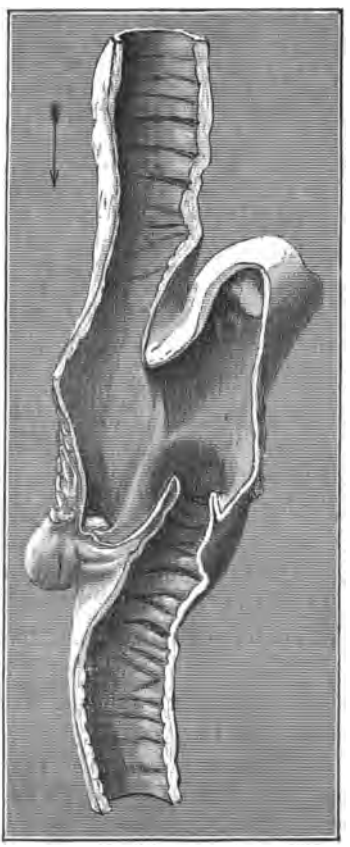


FIG. 4.—Dog. Lateral anastomosis. Senn's plates: 63 days. The arrow indicates the direction of the flow through the bowel. It will be noticed that the anastomosis still remains lateral. One blind end—that of the upper bowel—has contracted more than the other. ($\times \frac{2}{3}$.)

is more rapid. Indeed, the great advance of recent years in intestinal surgery is to be attributed to the experimental method.

A. Lateral anastomosis.—Various aids, in the shape of plates (Senn, Davis, Littlewood, &c.), bobbin (Mayo Robson), mechanical button (Murphy), are in use; or the operation can be carried out by suturing, and of sutures a large variety has been devised. Of these methods we will consider Senn's plates, Robson's bobbin, Murphy's button, and Halsted's method of suturing.

Senn's plates.—This method is so well known that it does not require description. The plates were cut to a size suitable for a dog's intestine, and the operation was carried out as now recommended by Senn, a continuous or interrupted suture being finally applied externally all round. The results of two of these operations are seen in Figs. 4 and 5. It will be noticed that the blind ends are short (much shorter, indeed, than at the time of operation), and that the anastomosis, which was originally quite lateral, has become end to end. Nature, in fact, in the short space of two months has made considerable progress, not only in the repair of the wound, but in the direction of the re-establishment of the original straight condition of the bowel. This process is further advanced in Fig. 5 than in Fig. 4. The opening, it will be seen, is of full size, the same size as the intestine.

Mayo Robson's bobbin.—This we used in one experiment, having had a bobbin made the right size for a dog. The result is shown in Fig. 6: the opening is small; it was, however, sufficient for its purpose, for the dog was quite well. It will be noticed that the valvulæ conniventes have entirely disappeared in the portion of the bowel above the anastomosis. We do not wish to infer too much from one experiment, and the successful employment of the bobbin combined with the continuous suture in its inventor's hands is well known to us. The small opening following the use of the bobbin is easily explained by the collapse of the bobbin shortly after the operation, and as only incisions have been made in the bowels, the edges come in contact, and heal except for the small opening.

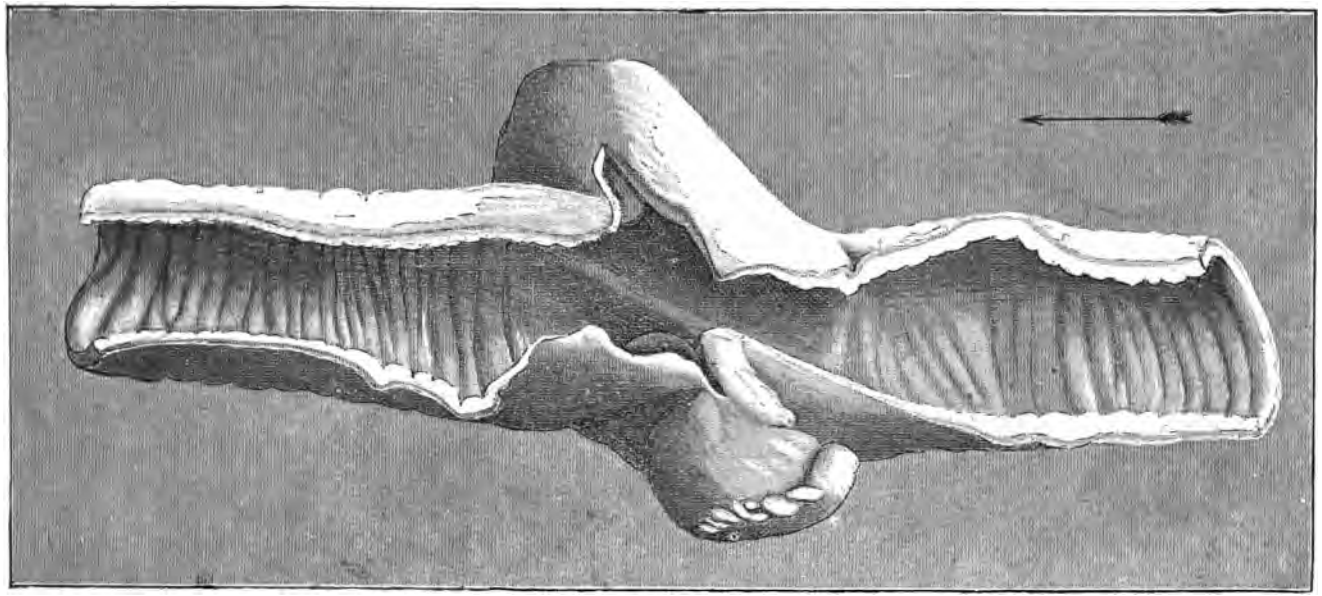


FIG. 5.—Dog. Lateral anastomosis. Senn's plates: 68 days. The arrow indicates the direction of the flow through the bowel. It will be noticed that although the anastomosis was originally completely lateral, the direction is now nearly straight; the two blind ends have become shorter. No trace of the plates. (Nat. size.)

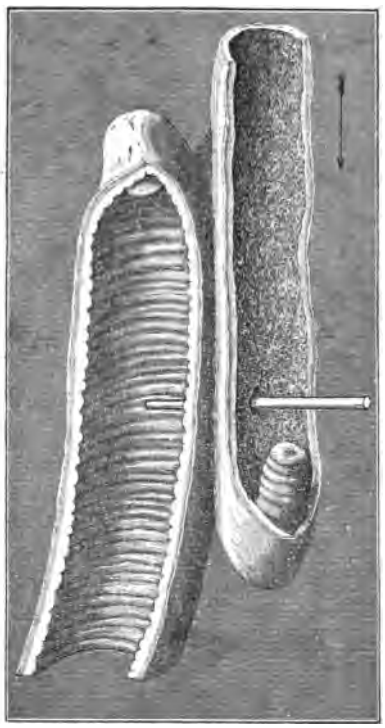


FIG. 6.—Dog. Lateral anastomosis. Mayo Robson's bobbin: 33 days. The arrow indicates the direction of the flow through the bowel. The anastomosis is quite lateral; the opening is small. A style is passed through opening. The blind end of upper bowel more shrunk than that of lower. (× ½.)

Murphy's button.—We tried this in one case, but the button we used, and which had been especially made for us for dogs, was somewhat too large, and this, therefore, cannot be considered a fair test; leakage occurred at the seat of union, and death ensued.

Pure suturing.—Finally, by this two anastomoses were made; the method of suturing used was Halsted's: both the animals did well. (See Figs. 43, 44, and 45.)

B. End to end junction.—Of the methods of end to end juncture, we tried Jessett's tubes, Paul's tube, pure

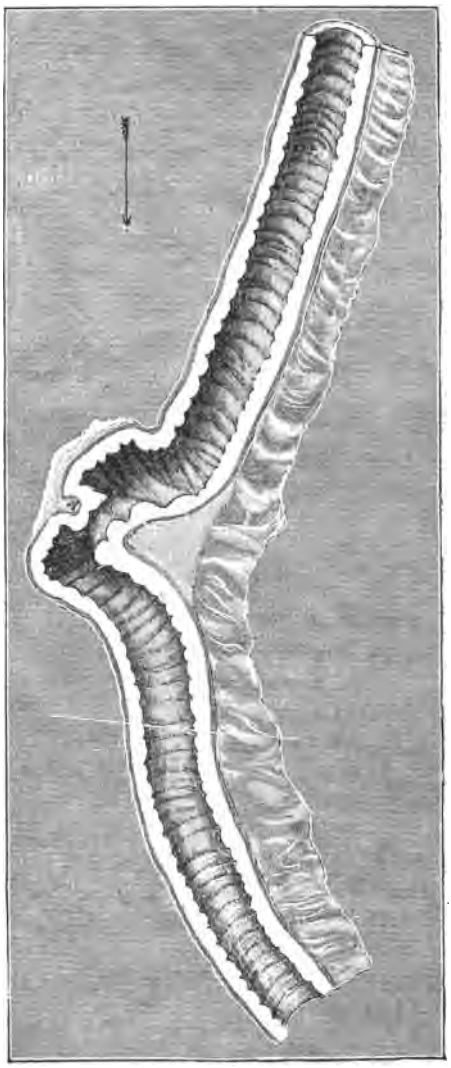


FIG. 7.—Dog. End to end suture: 11 days. The arrow indicates the direction of the flow through the bowel. The lumen of the bowel is considerably encroached upon at the point of suture; much more than in specimens obtained at a later date after operation. There is also a kink, due probably to excessive number of sutures at the mesenteric border, placed there to prevent leakage at a particular weak spot. Valvulæ conniventes alike above and below line of union. ($\times \frac{1}{2}$.)

suturing by the Czerny-Lembert method, and Maunsell's method.

Jessett's tubes seemed satisfactory, but with *Paul's* tube we completely failed to effect the requisite invagination of the bowel, and had to abandon the operation.

With *pure suturing* we did five operations by the Czerny-Lembert method, and two by Maunsell's method; these were all successful.

The five *Czerny-Lembert suturings* are seen in

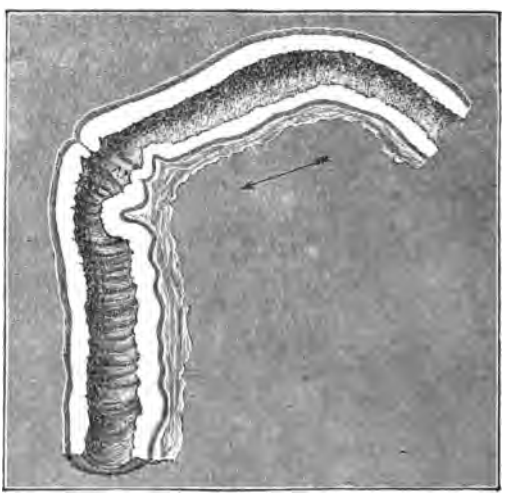


FIG. 8.—Dog. End to end suture: 18 days. The arrow indicates the course of the flow through the bowel. A slight ridge partly occludes lumen. Valvulæ conniventes seen below, but not above junction. (× ⅓.)

Figs. 7—11. They are all satisfactory, but they all have a circular ridge at the line of junction; this constistutes a diaphragm, which to some extent diminishes the lumen of the bowel.

In some of these cases the continuous suture was used for the mucous membrane, so that the operations were not all true Czerny-Lembert's.

There can be no doubt that the continuous suture is more rapid than the interrupted, but if the needles for

the latter are previously threaded, the difference in time need not be much; the objection, of course, to the con-

FIG. 9.

FIG. 10.

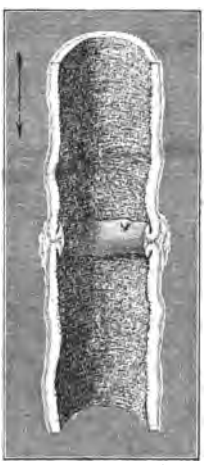


FIG. 9.—Dog. End to end suture: 21 days. The arrow indicates the direction of the flow through the bowel. The union is satisfactory; the lumen of the bowel is slightly encroached upon; there is some supporting tissue also externally. The two ridges formed by the incurving of the upper and lower portions of the bowel by the Lembert sutures are well seen. Valvulæ conniventes seen below, but not above line of junction. (× ½.)

FIG. 10.—Dog. End to end suture; 35 days. The arrow indicates the direction of the flow through the bowel. The union is satisfactory; the lumen of the bowel is slightly encroached upon; there is some supporting tissue also outside; the incurving of the coats by the outer row of (Lembert's) sutures is well seen. (× ½.)

tinuous suture is that it is not so easy to apply accurately, while with a Lembert stitch it is difficult to go wrong.

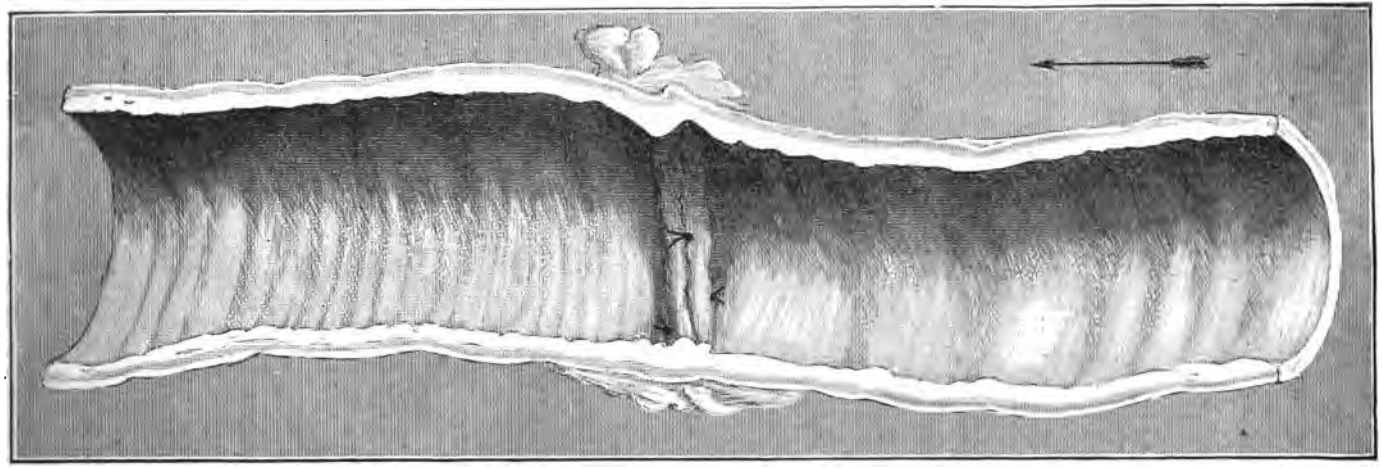


FIG. 11.—End to end sutures: 42 days. The arrow shows the direction of the flow through the bowel. The union is perfect; there are two slight ridges encroaching on the lumen, one on each side of the line of junction. Externally there is some organised lymph round the bowel. Three of the Lembert's stitches are seen working their way inwards towards the interior of the bowel. Valvulæ conniventes more marked below than above line of junction. (Nat. size.)

Lembert sutures of silk work inwards, and are discharged into the bowel, as was seen in some of the experiments, and is shown in Fig. 12.

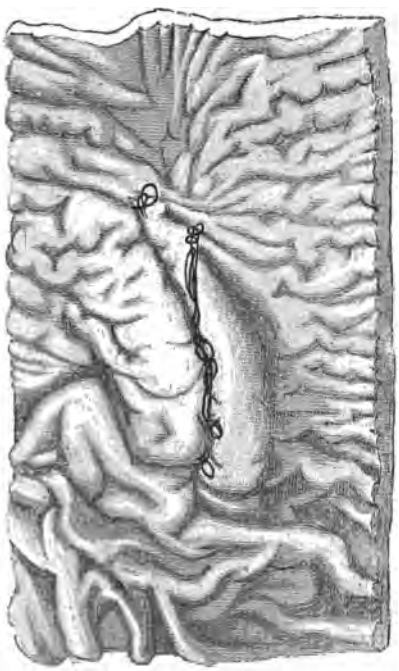


FIG. 12.—Reunion of a longitudinal incision in the large intestine of a woman æt. 35, for removal of fæcal concretion. Silk Lembert sutures were used. They are seen working out on the inner surface of the bowel six weeks after the operation. (Nat. size.)

Maunsell's operation, see Figs. 13, 14, and 26—29.—After this operation there is no diaphragm; indeed, it is quite difficult to recognise the line of circular junction: it contrasts markedly with the ridge which is seen at the site of the longitudinal incision which has been closed by Lembert sutures only.

C. Implantation of end of intestine into side.—This operation we performed twice, in both cases by pure

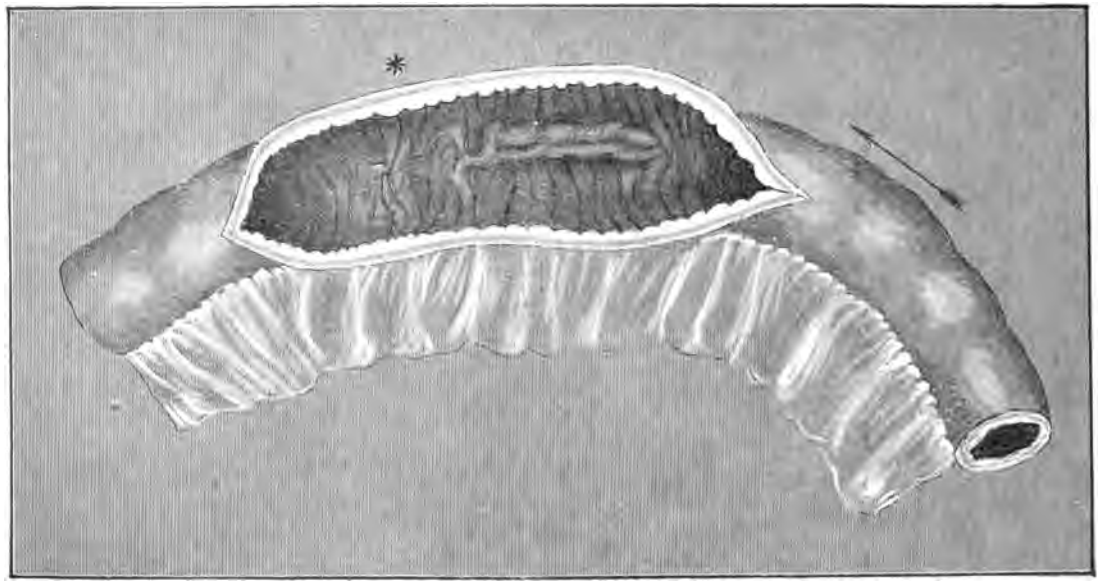


FIG. 13.—Dog. End to end union by Maunsell's method: 30 days. The arrow indicates direction of flow through the bowel; the asterisk the line of junction. This is extremely neat; there is scarcely any ridge within, and no new tissue without. This contrasts markedly with the line of union produced by Lembert sutures. The opening in the bowel made after death is lateral in order to show the median longitudinal incision, where there is now a considerable ridge produced by the Lembert sutures. Four Lembert's sutures were applied externally at the line of circular union (see abstract of experiments, No. 14). (Nat. size.)

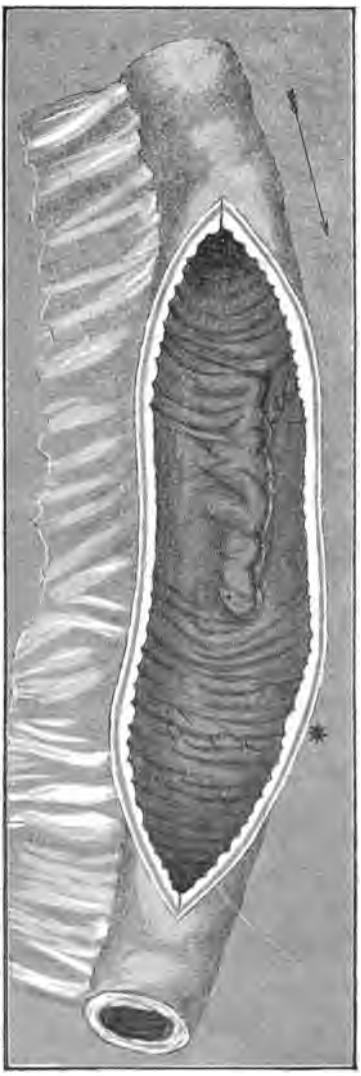


FIG. 14.—Dog. End to end suture by Maunsell's method: 78 days. The arrow indicates direction of flow through bowel. The bowel is opened at the side to show the median ridge caused by the suturing of the longitudinal incision with only Lembert stitches. At this level the bowel is now of smaller calibre than above or below (3 mm. less in circumference). At this level, too, the valvulæ conniventes are absent. The circular junction marked by an asterisk is almost invisible, there being no encroachment on the lumen of the gut. Several Lembert sutures were applied externally at line of circular union (see abstract of experiments, No. 15). ($\times \frac{3}{4}$.)

suturing ; they both did well. Vertical implantation is a form of short circuiting; the choice between it and lateral anastomosis must be decided by the individualities of the case, such as the length of bowel available : in lateral anastomosis a complete diversion of the chyme can

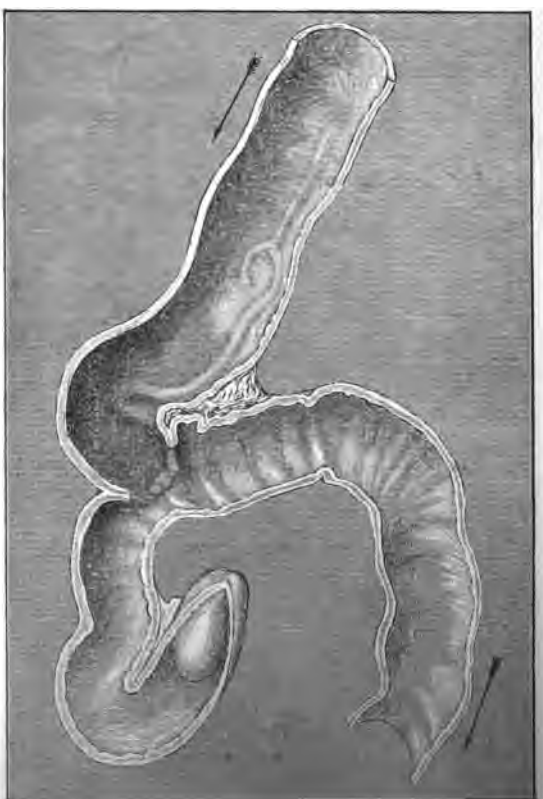


FIG. 15.—Dog. Implantation of upper into lower part. The arrow indicates the direction of the flow of the contents of the bowel. It will be noticed that although the anastomosis was vertical, yet the direction of the flow is now nearly straight. The blind end is small in comparison with that in Fig. 16, in which the anastomosis was made in the reverse way. Specimen obtained after 84 [illegible].

be effected if desired by the division of the bowel, and the inversion and suturing of the ends.

Fig. 15 shows implantation of the upper end of the divided bowel into the side of the lower; Fig. 16 shows

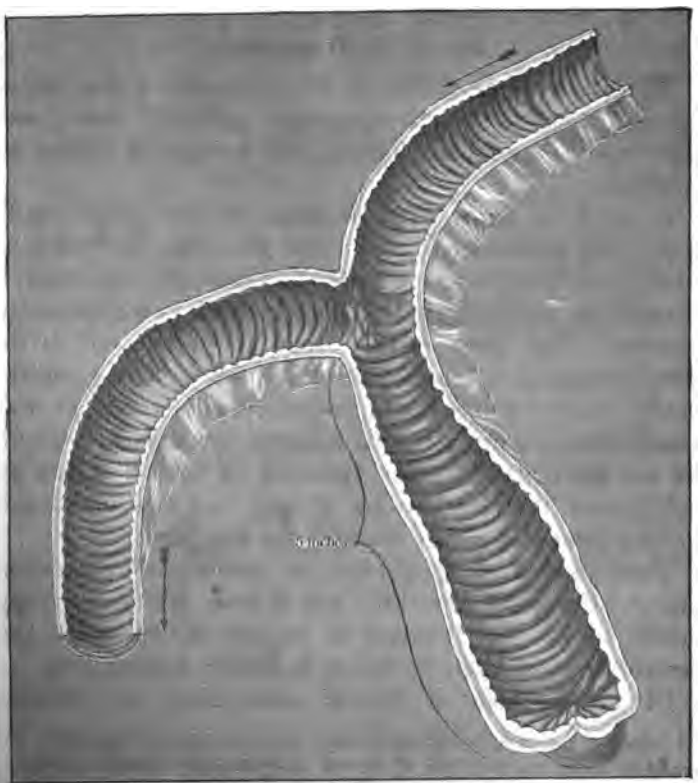


FIG. 16.—Dog. Lower bowel has been implanted into upper. The arrows indicate direction of flow through bowel. The blind end is five inches long, the same length as originally; it is distended, its wall hypertrophied, as are also the valvulæ conniventes. Specimen obtained in 126 days. Note distension of the blind end in connection with the direction of peristalsis. ($\times \frac{1}{4}$.)

[illegible] of the lower end of the divided bowel into [illegible] of the upper.

[illegible] intestinal clamps, although several

ingenious forms have been devised, we have not seen any reason to be dissatisfied with the use of a piece of rubber tube passed through a hole in the mesentery, and tied with a single hitch; the bowel is thus perfectly occluded and not injured.

Choice of Operation.

There are three main conditions under which these operations may be required, namely, artificial anus (from any cause), gangrenous hernia, and malignant disease of intestine.

A. *Artificial anus.*—The condition of fæcal fistula often undergoes spontaneous cure, and this must be borne in mind before deciding to operate. The method of Dupuytren of removing the spur with the enterotome is now apparently obsolete. The next method that suggests itself is an extra-peritoneal operation, as described by Greig Smith. Lastly, there remains the intra-peritoneal methods, as first practised successfully in this country by Makins. There are two methods of uniting two ends of bowel; either by end to end suturing, or by closing the two ends, and making lateral openings between the two parts.

These operations should not, of course, be undertaken except after some practice; but if from any cause they should have to be, it seems to us that the operation the surgeon is least likely to fail in is lateral anastomosis.[1]

Of the methods of lateral anastomosis we consider

[1] "One of the most important lessons that our experimental work has taught us is that the principle of *lateral approximation* should be used in every instance in which it can be applied. In itself it is a very simple and easy operation, and can generally be completed in from ten to fifteen minutes. In not a single case did we lose a dog on which we had performed a lateral approximation of two loops of the small intestine with or without resection, except when we experimented with the cut ends after resection. Such has been, to a great extent, the experience of all operators. The principle of lateral approximation may be carried out almost *ad infinitum*. If two sections of the small intestine are to be joined, a lateral approximation should be made; if the small intestine and the stomach are to be brought together, the union should be by lateral approximation; if the small and large intestine are the parts to be dealt with, still a lateral approximation is the operation of choice: indeed, this method should be practised if any of the other hollow

Halsted's superior to Senn's, and indeed to all methods in which plates, bobbins, or other mechanical aids are relied on. Figs. 16—22 will remind the reader of the details of Halsted's operation.

One objection to all mechanical aids is that it is possible that the surgeon will not have the right sized plate or bobbin at hand; it is often claimed that these shorten the time of operation, but if Lembert sutures are placed round the plates, the time is not much shortened; further, we are aware of cases in which, with plates and buttons, leakage and death have occurred. Halsted's operation need not take long if the following details are attended to:—more than sufficient needles should be ready threaded: the needle we prefer is No. 8 straw needle; the silk should be sufficiently thick not to cut the intestine; No. 0 silk is the size we have used.

We found it much better to have two needles on each suture, one at each end; by this means the operation can be more rapidly performed, for it is much easier for the surgeon to pass all the needles in the same way, than to have to pass each needle back again in the reverse direction; further, if one needle is passed only half through at first, it acts as a splint, and steadies the bowel for the next needle, and so on. In some experiments we united with a few stitches the two cut edges of mucous membrane.

Figs. 23 and 24 show the result of a Halsted operation on the human subject by one of us. An account of another of our cases will be found in the 'Clinical Society's Transactions' for 1894.

Of the methods of end to end anastomosis we again

viscera are to be united. So thoroughly are we convinced of the greater safety of this operation over any of the other methods, that with our present experience we would not dare to attempt an end to end approximation or an invagination on the human subject in cases in which we had the choice. Our mortality from the end to end approximation after resection amount to more than 30 per cent. Brokaw in his experiments had a mortality of 50 per cent., and other experimenters have shown no better results, whatever method was employed."—Ashton and Baldy, *Experimental Studies in Intestinal Surgery*, 'Medical News' (U.S.A.), Feb. 28th, 1891.

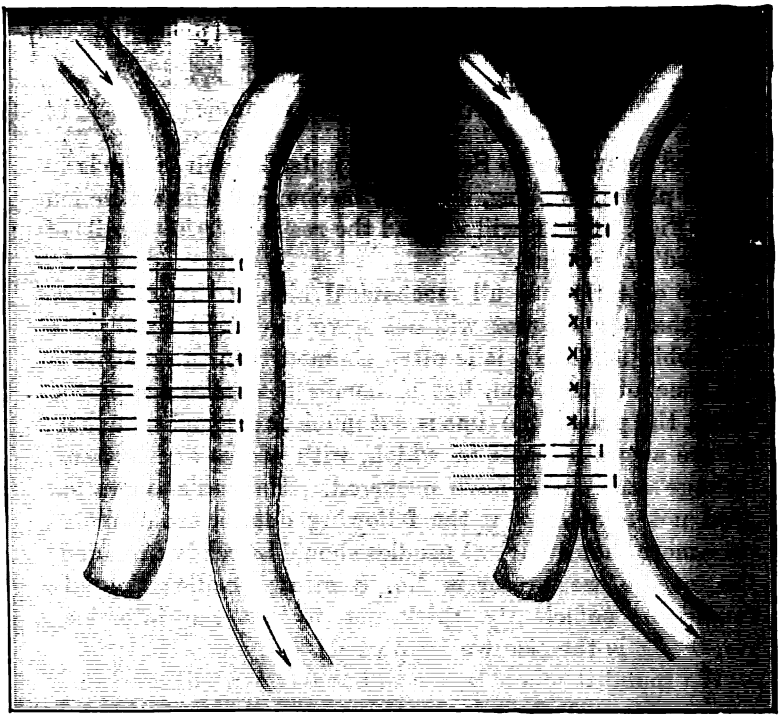


FIG. 17.

FIG. 18.

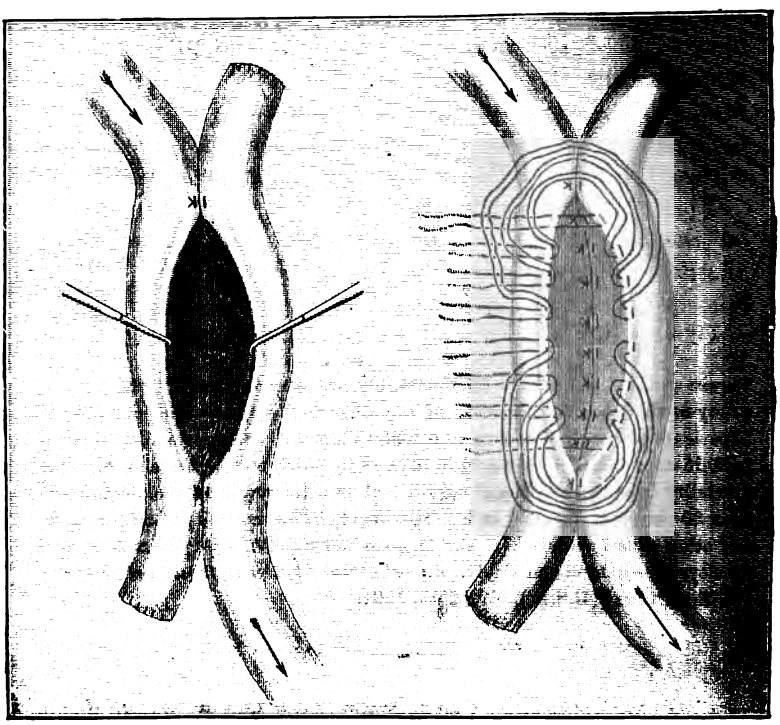


FIG. 19.

FIG. 20.

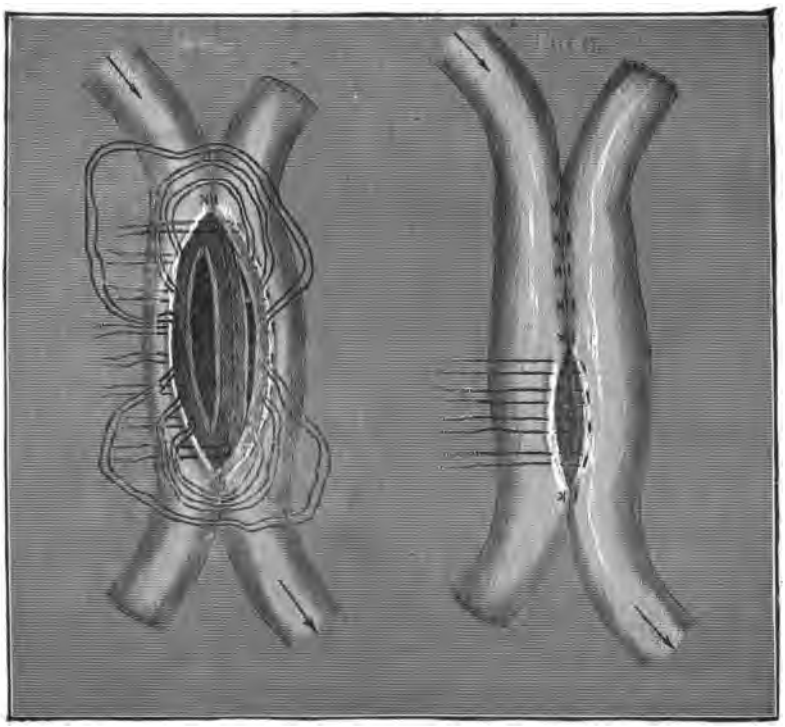


FIG. 21. FIG. 22.

HALSTED'S OPERATION (after Halsted).

FIG. 17.—1*st stage.* Posterior row of sutures applied.
FIG. 18.—2*nd stage.* Posterior row of sutures tied. Lateral sutures applied but not tied.
FIG. 19.—3*rd stage.* Posterior and lateral sutures tied.
FIG. 20.—4*th stage.* Anterior row of sutures applied and drawn aside. Intestine not yet incised.
FIG. 21.—5*th stage.* Intestine incised.
FIG. 22.—6*th stage.* Anterior row of sutures. Four tied and four not tied.

prefer simple suturing to the use of any form of supporting apparatus such as recommended by Paul, Jessett, and others.

Of these mechanical devices, some (as Robson's bobbin) are not attached to the bowel; these seem to us much better than those which are either attached by sutures to the bowel, as Senn's plates and Paul's tube, or, as in the

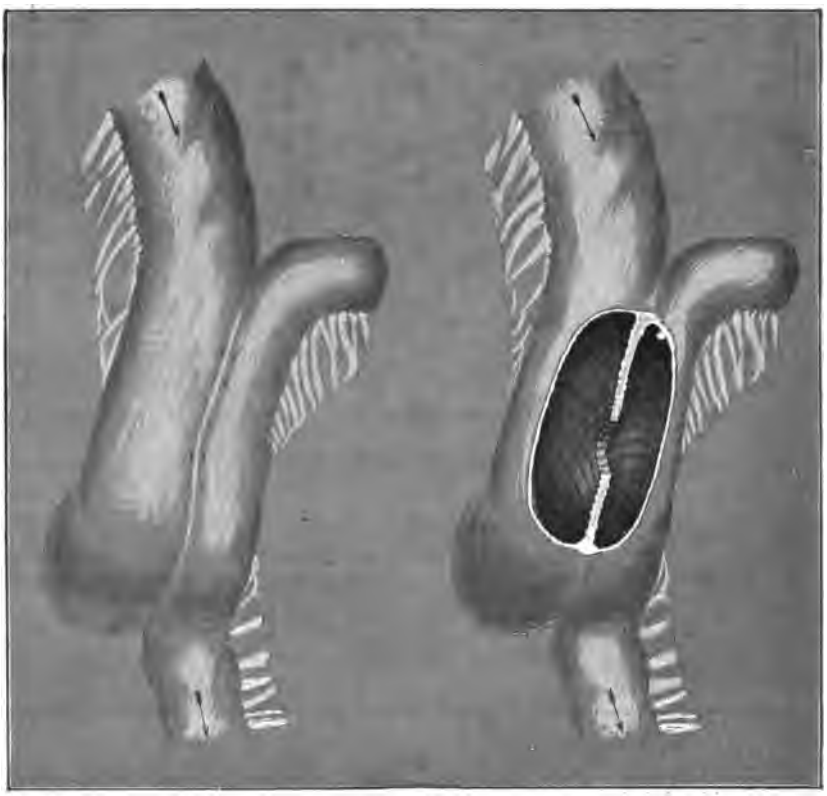


FIG. 23. FIG. 24.

Gangrenous hernia treated by Halsted's operation. Woman æt. 56. Small intestine strangulated in an umbilical hernia. Patient was the subject of huge fibroma of ovary, which caused intestinal obstruction and death fifth day after operation. The union was complete, and withstood high water pressure. Specimen in St. Thomas's Hospital Museum. (× ½.)

case of Murphy's button, are attached by compression to the bowel, the included portion of which is to become gangrenous.

The results of direct suture by the Czerny-Lembert method are shown in the accompanying drawings. Most care has to be taken at the mesenteric border, and here the

sutures should be applied first; the eversion of the mucous membrane in the small intestine is so great that the inner row of stitches merely results in the apposition of mucous membrane to mucous membrane, and the integrity of the junction depends solely on the Lembert sutures (see Fig. 25).

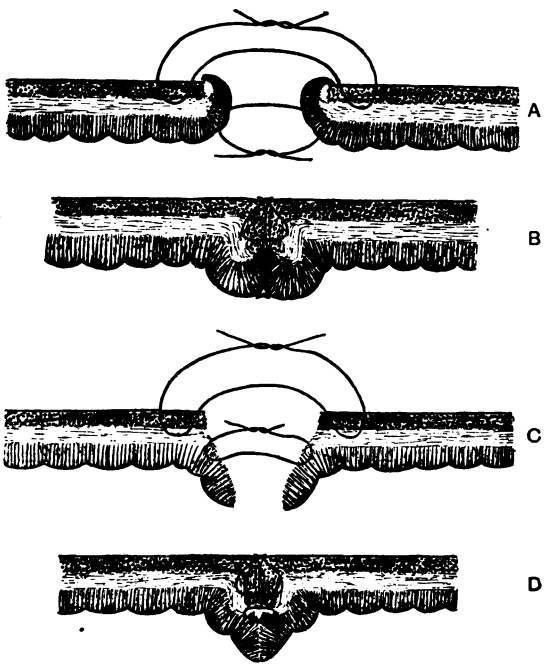


FIG. 25.—A shows the way in which intestine is often brought together by the Czerny-Lembert method; mucous surface being brought to mucous surface. B shows the sutures in fig. A tightened. C shows sutures applied so as to bring raw surface of mucous membrane to raw surface of mucous membrane—a method which can be often practised in the large intestine. D the sutures shown in fig. C tightened and the bowel brought together.

The result of this is the ridge which remains at the line of junction, sometimes seriously contracting the lumen. The temptation is to put in a second or even third row of Lembert sutures, and thus to run the risk of occluding

the bowel. The employment of the bobbin prevents this form of obstruction at least for the short time it retains its shape. The eversion of the mucous membrane does not occur to the same extent in the large intestine, and therefore the raw surface of the mucous membranes and of the muscular coat can be brought into apposition by the inner row of stitches.

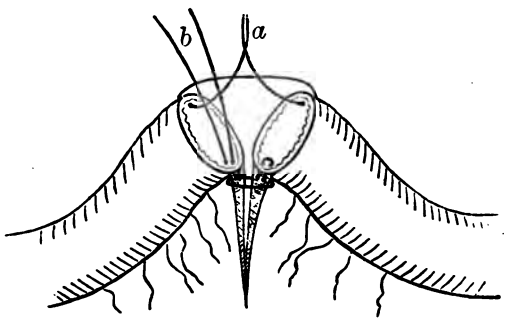


FIG. 26.—Maunsell's operation. 1st stage. Sutures of approximation in free (*a*) and mesenteric (*b*) borders passed, but neither tied. This and the three following figures are by Stanley Boyd (after Maunsell).

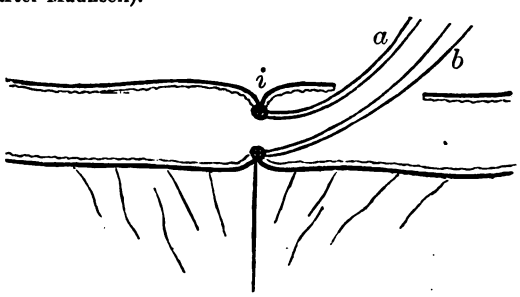


FIG. 27.—Maunsell's operation. 2nd stage. Sutures of approximation tied, and their ends (*a* and *b*) passed through a cut in the free margin. *i*. Line of section.

This objection does not apply to Maunsell's method (Figs. 26—29); it causes very perfect union of the two parts of bowel; raw surface of mucous membrane is brought to the same, muscular coat to muscular coat, and peritoneum to peritoneum. The result is an almost per-

fect union, the site of which has to be looked for to be detected (see Figs. 13 and 14). The circular union con-

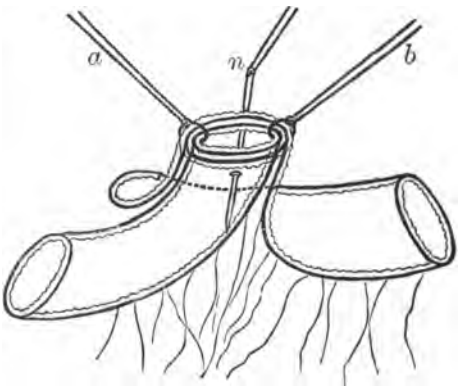


FIG. 28.—Maunsell's operation. 3rd stage. Traction has been made on *a* and *b*, producing an invagination, the layers of which are visible (dark lines = serosa, wavy = mucosa). *n*. A needle passed through all four layers of bowel.

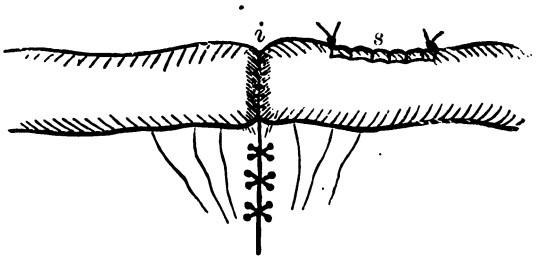


FIG. 29.—Maunsell's operation. 4th stage. Invagination reduced. *i*. Line of union. *s*. Wound in free margin closed by continuous suture. Mesentery has been sutured by interrupted stitches.

trasts markedly with the union by Lembert sutures of the longitudinal incision (Figs. 30 and 31). A possible objection to Maunsell's operation is that the sutures pass completely through the bowel, and may thus lead to leakage. In both our experiments we thought it necessary to place additional Lembert sutures at line of circular union (see abstract of experiments).

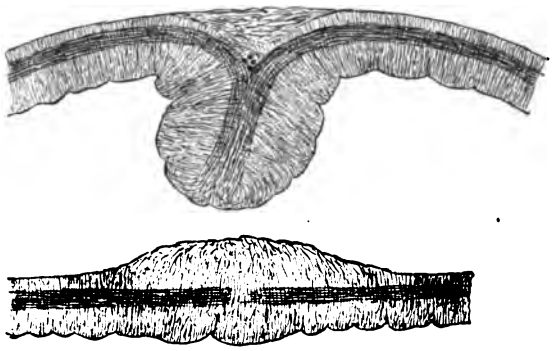


FIG. 30.—From the intestine of a dog on which thirty days previously a Maunsell's operation for end to end junction had been performed (see Fig. 13). The upper figure shows a transverse section of the healed longitudinal incision which was closed by Lembert's sutures, one of which is seen *in situ*. The lower figure is a section of the healed circular line of union. The figures show well the internal ridge formed by the Lembert sutures alone, as contrasted with the absence of obstruction to the bowel in the part united by Maunsell's method of suturing. (× 4.)

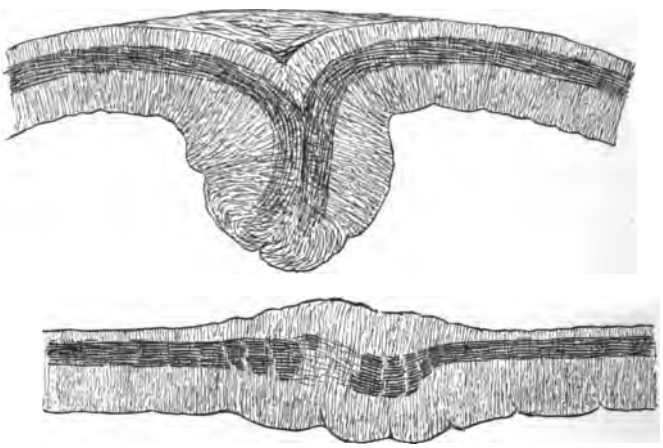


FIG. 31.—From the intestine of a dog on which seventy-three days previously a Maunsell's operation for end to end junction had been performed (see Fig. 14). The upper figure shows a transverse section of the healed longitudinal incision which was closed by Lembert sutures. The lower figure is a section of the healed circular line of union. The figures show well the ridge formed by the Lembert sutures alone, as contrasted with the absence of obstruction to the bowel in the part united by Maunsell's method of suturing, although additional Lembert sutures were used. (× 4.)

B. With regard to *gangrenous hernia,* the present tendency of surgery seems to be towards excision and primary suture, and the remarks already made will apply to this.

C. As to the treatment of *malignant disease of bowel,* if the growth can be removed, it of course should be, and the intestine will be either at once or subsequently united. As new growth generally occurs in the great bowel, and the small intestine is only rarely affected, the question will be one of union of great bowel either to itself or with small bowel. In the case of great bowel to great bowel there will probably not be sufficient length of intestine left after excision of growth to do a lateral anastomosis or Maunsell's method, and therefore the surgeon is restricted to another form of end to end junction, or to an implantation.

The following figures (32—36) illustrate the treatment of a case of malignant disease of the sigmoid by one of us.

In the case of uniting small bowel to large bowel (as after removal of a growth in the cæcum) the choice of operation is possibly wider, and lateral anastomosis and Maunsell's operation are practicable, or an implantation can be done; this latter would seem proper, owing to the difference in size of the two portions of bowel. It is probable that the safest and easiest operation—where possible—would be lateral anastomosis by Halsted's method; where not possible, as sometimes when junction is made with the rectum, a large Murphy's button could be used, as in a successful case under the care of one of us, in which the sigmoid was opened into the rectum for the cure of artificial anus. Where the growth cannot be removed and obstruction exists there are only two things the surgeon can do—either to make an artificial anus above, or to make an artificial opening between the bowel above the growth and that below. By the latter method there will be a choice between (*a*) simple lateral anastomosis, (*b*) lateral anastomosis after division of the bowel above

the tumour, and (*c*) implantation after division of the bowel. The best operation will depend partly on the case and partly on the surgeon, but the last two methods

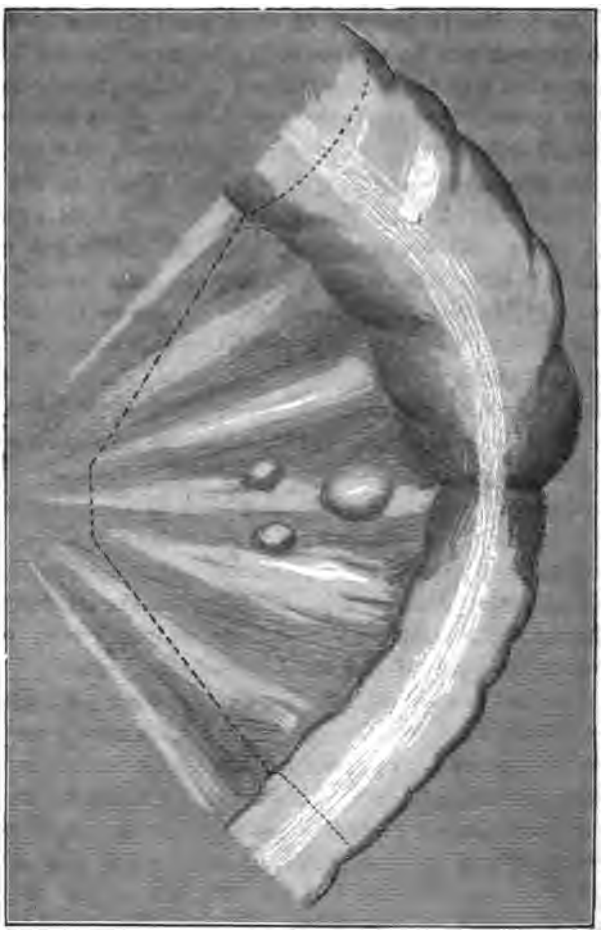


FIG. 32.—Drawing shows a case of carcinoma of sigmoid flexure with glands in mesentery. Patient a woman æt. 48. The dotted line shows the line of section by which the tumour was removed. There was considerable obstruction and much distension of abdomen. Both ends of bowel brought out and subsequently reunited (see Figs. 33 and 34). ($\times \frac{1}{4}$.)

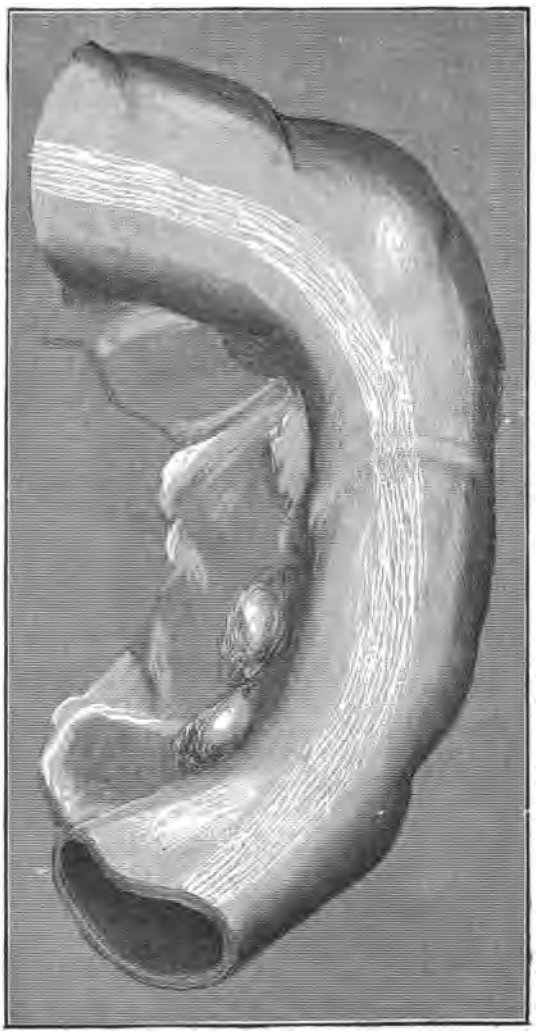


FIG. 33.—Two months after reunion of sigmoid (as mentioned in description of Fig. 32) patient died, and specimen figured obtained. It will be noticed that the direct line of the bowel was re-established. Only a line marks the site of reunion. ($\times \frac{1}{4}$.)

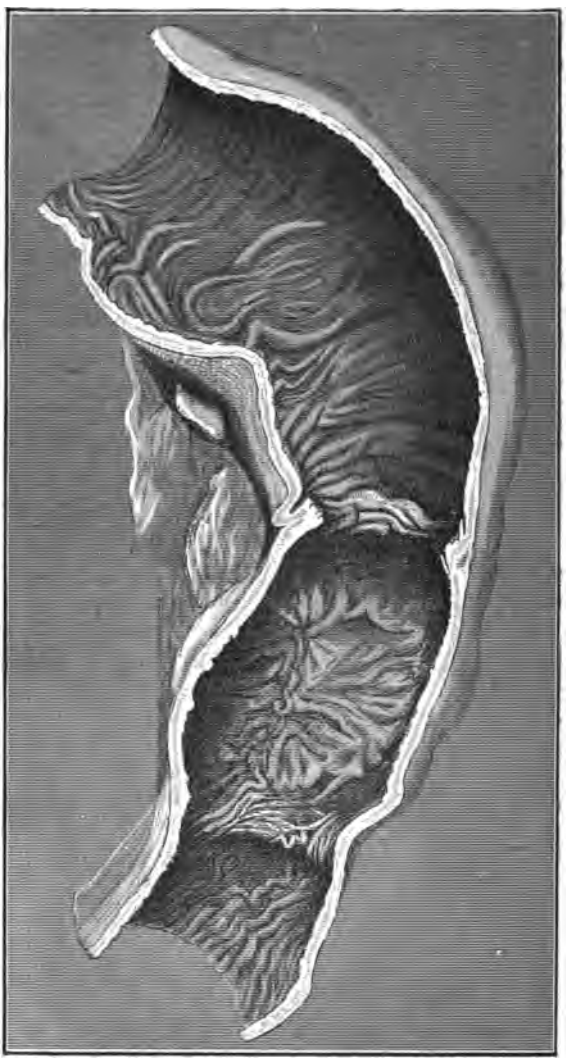


FIG. 34.—Internal view of bowel shown in Fig. 33. A ridge marks the line of union, the suturing being Czerny-Lembert. The raw surface of mucous membrane was stitched to the same (not mucous surface to mucous surface). ($\times \frac{3}{4}$.)

will have the advantage of preventing the passage of fæces over the growth; this diverting of the fæces not

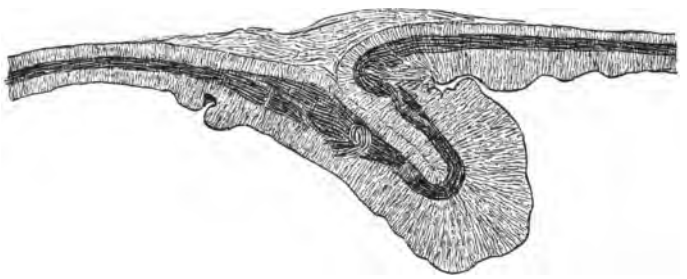


FIG. 35.—From a woman who had undergone excision of a portion of the sigmoid flexure for carcinoma (see Figs. 32—34). The divided ends were subsequently reunited. The figure shows a section of the circular line of union and of the diaphragm caused by the Czerny-Lembert suturing. (×3.)

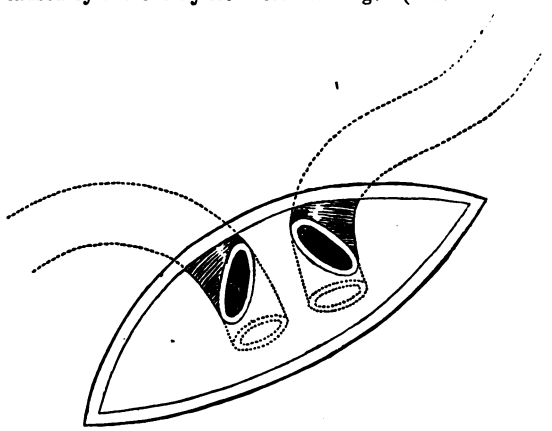


FIG. 36.—Diagram illustrating oblique section of bowel in end to end anastomosis for the cure of artificial anus. The advantages of the oblique section are (1) larger lumen to suture, and (2) angle at the mesenteric border is lessened or abolished. The method was adopted in the operation for the cure of artificial anus in the case illustrated in Figs. 32—35. This section might not be advisable in a recent case, or at least care should be taken not to interfere with the blood supply to the bowels.

merely stops any irritation of the growth by them, but also possibly causes some retardation of the rate of

growth comparable to the atrophy of bowel which occurs in short-circuiting experiments even where the passage of the chyme is not completely stopped. Implantation can probably only be performed when small intestine forms the upper part of the junction. An interesting case of implantation for artificial anus after gangrenous hernia is related by Davies-Colley in the 'Guy's Hospital Reports;' here the ileum was implanted into the ascending colon.

There is a very real danger in performing an excision and reunion operation primarily in cases in which there is even a minor degree of obstruction. The pent-up liquid fæces may pass in considerable quantity over the fresh wound, and this, together with the muscular action of the bowel, make most unfavourable conditions for union.

The alternative to excision and immediate reunion is excision and bringing out the ends to make an artificial anus. In many of these cases an acute obstruction supervenes on a long-continued chronic obstruction, and these cases are not suitable for the temporary drainage of the bowel, such as can be applied for a few hours in cases of strangulated hernia with much distension, as recommended by Greig Smith.

D. With respect to *intussusception* the question of treatment is too large to be discussed here, but it is interesting to note the resemblance between one of Nature's methods of spontaneous cure, namely, by the sloughing of the intussusceptum, Barker's method of operation by incising the bowel and amputating the intussusceptum, and finally Maunsell's operation for reunion, in which an intussusception is first produced. If a total excision is thought advisable, the surgeon must decide according to the case between lateral and end to end anastomosis.

Volvulus, especially that chronic form of it which occurs in the omega loop of the sigmoid flexure, occasionally requires operation: hitherto relief has generally been sought by an artificial anus; but it suggests itself that

a better result might be obtained by a large Halsted lateral anastomosis between the two lower arms of the large loop—in some cases it might be advisable to make in addition an artificial anus in the loop (Fig. 37). The advantage of the anastomosis is that the loop might be expected to atrophy from disuse—in fact, by precisely the reverse process to that which had produced it.

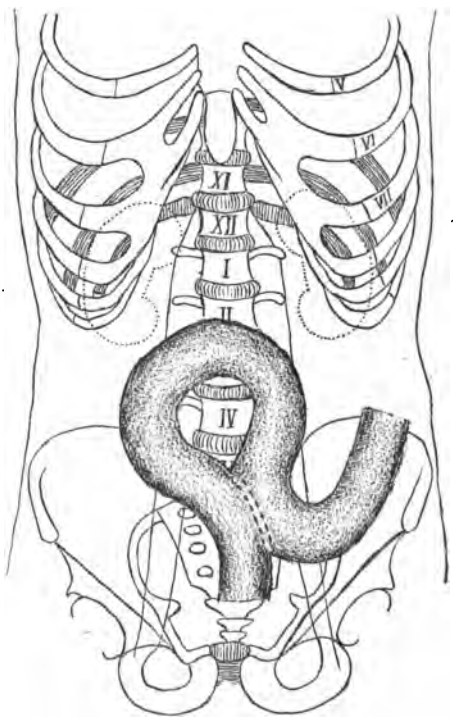


FIG. 37.—Figure modified from drawing (fig. 9) in article by von Samson on variations of the sigmoid flexure in Langenbeck's 'Archiv für klin. Chirurgie,' vol. xliv, p. 386 (1892). In the modified drawing the enlarged and distended sigmoid flexure is represented as united at the base of the omega by a Halsted's anastomosis. It is suggested that the bowel should be thus short-circuited, and that the loop would then gradually undergo atrophy from partial disuse.

II. Gastro-enterostomy.

It is still a moot point how best to perform gastro-jejunostomy for the relief of pyloric obstruction due to malignant disease or other cause ; even if pylorectomy is thought advisable it may be best to perform gastro-jejunostomy first. There has been some discussion as to whether the intestine should be attached to the front or to the back of the stomach, also as to what method of attachment should be employed, also as to whether the direction of the jejunum should be from right to left or from left to right.

As surgeons are not frequently called on to perform this operation, perhaps the most obvious mode is best : this consists in finding the commencing part of the jejunum and bringing it across the omentum to the front of the stomach and uniting it there by Halsted's operation ; an opening of considerable size can be made (Figs. 38—40).

In one of the gastro-jejunostomies the portion of bowel between the pylorus and the artificial opening was dilated and was much larger than the jejunum beyond (Fig. 39). It is probable that some of the food which passed through the pylorus re-entered the stomach at the new opening, and thus passed round and round : in this way the duodenum would to some extent be acting the same part mechanically as the stomach : any obstruction to the passage of chyme at the seat of opening would also account for the distension of the bowel above it.

The part of the stomach that lies naturally nearest the first part of the jejunum is its posterior surface, and the least disturbance of relations would be obtained by attaching these together : this method has been strongly advocated by Paul, who uses an ivory ring to effect the attachment; Fig. 41 illustrates the result of this operation in a dog. Paul further claims for this method that the opening will not spontaneously close, as seemed to have happened in several cases in which the anterior operation was performed with plates or bobbins. With Halsted's

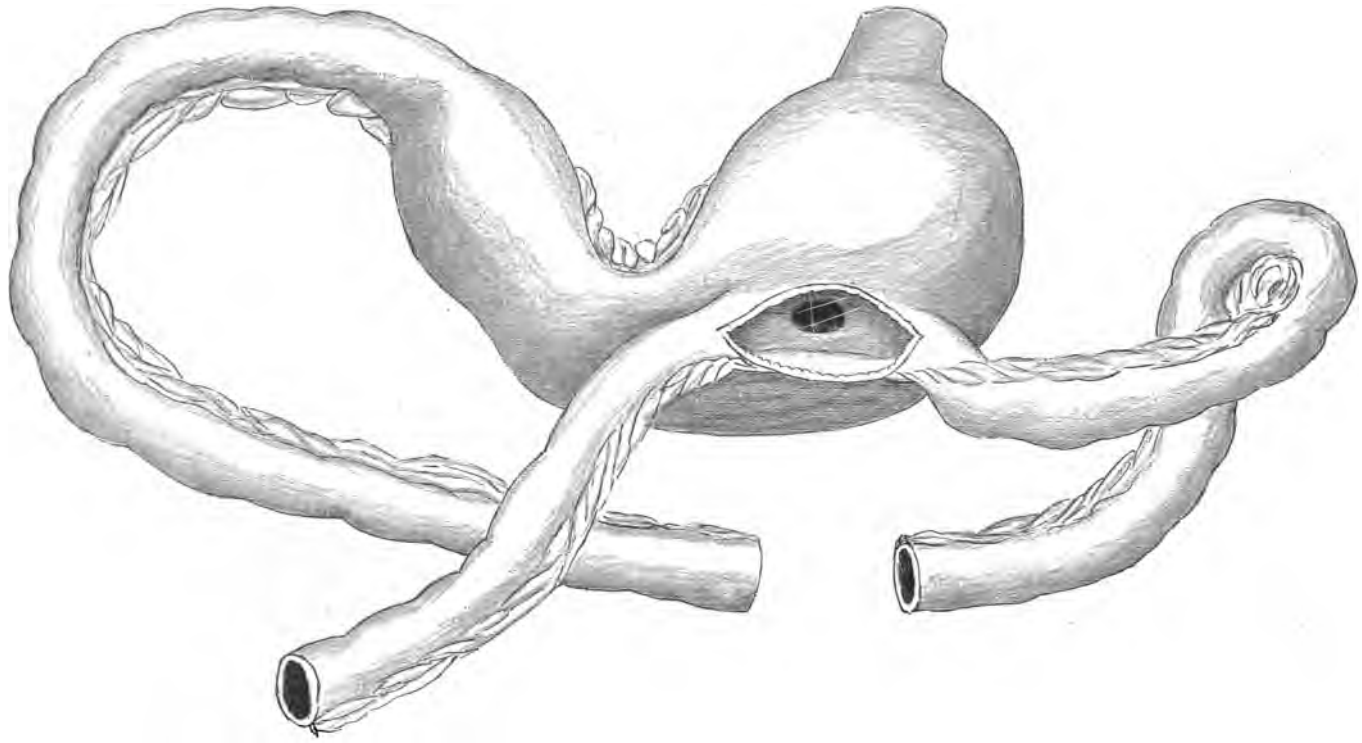


FIG. 38.—The jejunum was attached to the anterior surface of the stomach and opened into by Halsted's method of suturing; a portion of the mucous membrane of the stomach was cut away. Animal killed on 63rd day. The pylorus is not distended, nor is the bowel between it and the artificial opening (as in Fig. 39). The anastomotic opening has contracted to about half its original dimensions; it is now 10 × 7 mm. The opening in the duodenum was 27 inches below the pylorus. ($\frac{2}{3}$ scale.)

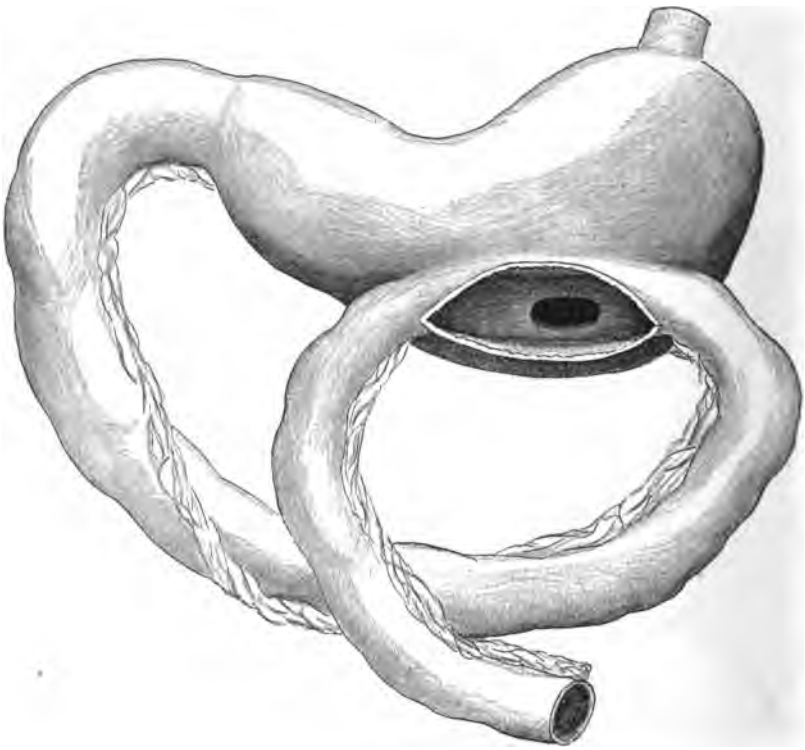


FIG. 39.—The jejunum was attached to and connected with the stomach (anterior surface) by an opening 42 mm. (1¾ inches) long. The opening now is 18 × 7 mm. Halsted's method of suturing was used. Dog lost flesh; killed 70th day. The duodenum and portion of jejunum between stomach and artificial opening is distended; that beyond is smaller than normal. The explanation of this distension of the duodenum is probably that the portion of food which passed through the pylorus in part re-entered the stomach at the artificial opening, thus going round and round. (× ⅓.)

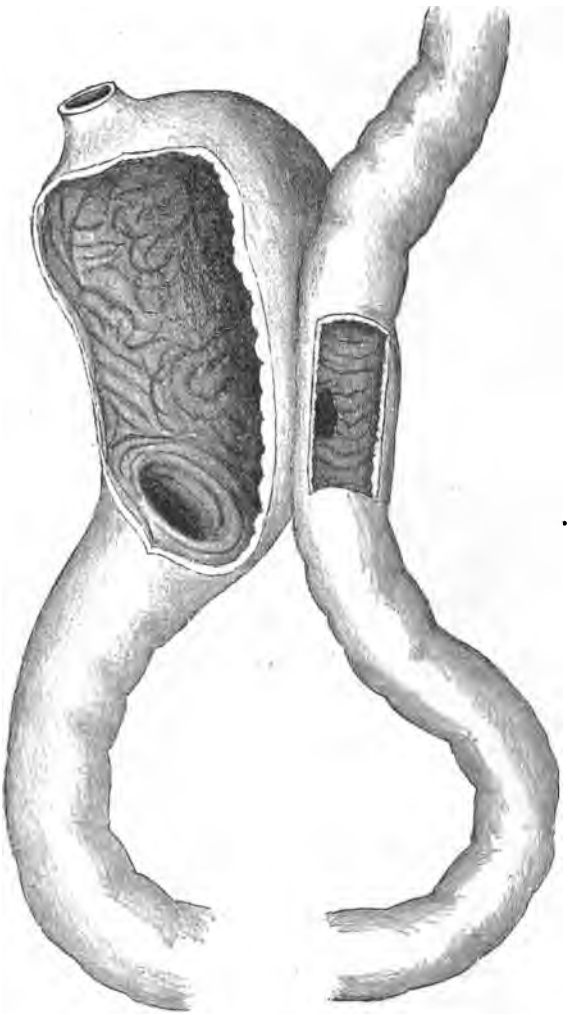


FIG. 40.—Stomach and portion of ileum united; the ileum had been brought up to the anterior surface of the stomach, sutured, and opened into it by Halsted's method. The dog did well, and was killed the 76th day. The artificial opening is free. In this case it will be noticed that the ileum was attached from right to left. (× ⅓.)

operation, however, the size of the opening is within the surgeon's control. Paul attaches great importance to the

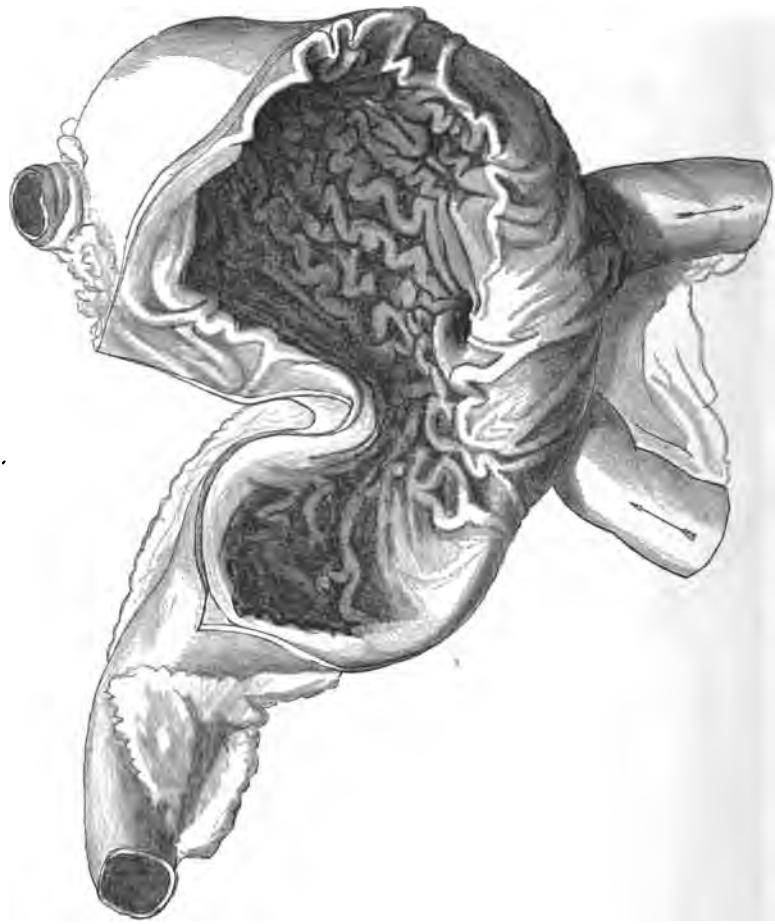


FIG. 41.—Stomach and portion of jejunum of a dog in which gastro-jejunostomy by Paul's method had been performed 97 days previously. The opening is amply sufficient. There does not appear to have been any contraction. The opening is on the posterior surface of the stomach. (Scale $\frac{2}{3}$.)

strangulation and death of a small portion of the walls of stomach and intestine which occurs in his operation as

preventing the tendency to contract. In Figs. 38—40 are shown gastro-jejunostomies by Halsted's method in which a portion of the prolapsing mucous membrane of the stomach was excised. It is obvious that by any method a portion of the whole thickness of the stomach wall can be excised if that is thought essential to ensure the opening remaining patent.

It is probable that it makes no material difference in which direction the jejunum is attached to the stomach, that is to say, whether from right to left or left to right; according to physiology it should be from left to right, for the direction of the peripheral current of food in the stomach is said to be towards the pylorus.

For pyloric obstruction not due to malignant disease it may be advisable to merely divide a band or to perform pyloroplasty, or in some cases gastro-jejunostomy.

In grafting the jejunum on to the front wall of the stomach it is obvious that care must be taken to avoid any twisting of the mesentery or any kinking of the intestine. Gastro-jejunostomy can also be performed by means of Maunsell's operation, as can also the reunion after pylorectomy—as described and figured by Wiggin after Maunsell. It suggests itself that it would be better for the artificial opening to be in what is ordinarily the most dependent part of the stomach; but the muscular contraction of the stomach would probably empty the organ wherever the artificial opening is situate. The pylorus itself is not dependent.

III. Physiological Observations.

1. In *lateral anastomosis* it is interesting to notice that the blind ends become shorter (see Figs. 4 and 5)—atrophy in fact—and would probably in time entirely disappear; further, the lateral opening after a time ceases to be lateral and tends to become more and more terminal or in the axis of the bowel; thus in time it is to be expected that the normal condition would be restored and all trace of

the operation lost. It would seem that the less specialised a part is the more perfect is repair.

2. In *short-circuiting* experiments the loop which is partly excluded diminishes markedly, both in length and diameter: in one case in 78 days it had diminished in length from 7 to 3½ inches, that is to say reduced to 46 per cent. of its original length. The same atrophy is observed when the large intestine is totally excluded by an artificial anus in the ileum. This would seem to

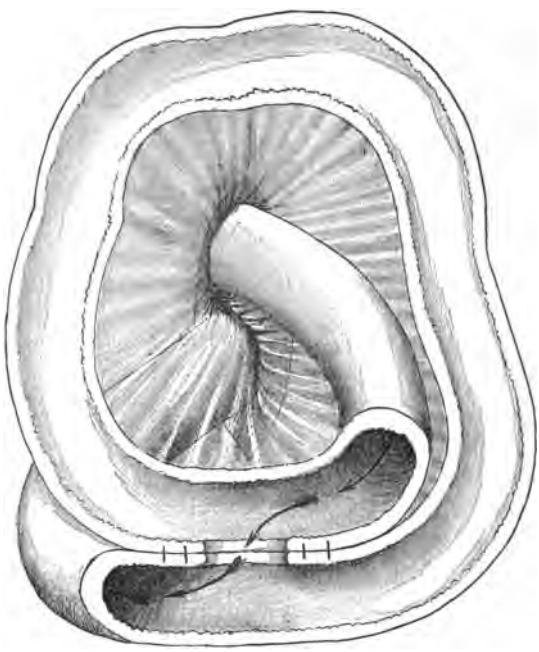


FIG. 42.—Diagram to illustrate the mode in which the short-circuiting of the small intestine of the dog was performed by Halsted's operation of lateral anastomosis. See Figs. 43—45.

show that the continued discharge of function is a necessary condition to the continued existence of the structure of the part—in fact, that without physiology there could be no anatomy. Fig. 42 illustrates the plan

adopted in these short-circuiting experiments. Figs. 43—45 are drawings of short-circuited portions of intestine for from 78 to 165 days after operation.

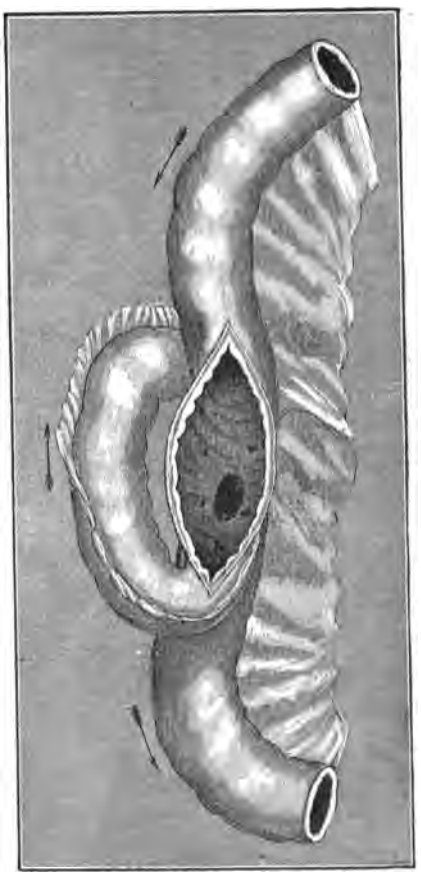


FIG. 43.—Small intestine was short-circuited by Halsted's method. Specimen obtained after 78 days. The excluded loop is seen to be diminished in diameter; it has also become reduced in length from 18 cm. to 8 cm. The artificial opening measures 7 mm. in its long diameter. The Halsted sutures are seen to be working their way to the interior of the bowel. In the abdomen the excluded loop lay transverse to the long axis of the bowel made by the upper and lower ends. ($\times \frac{2}{3}$.)

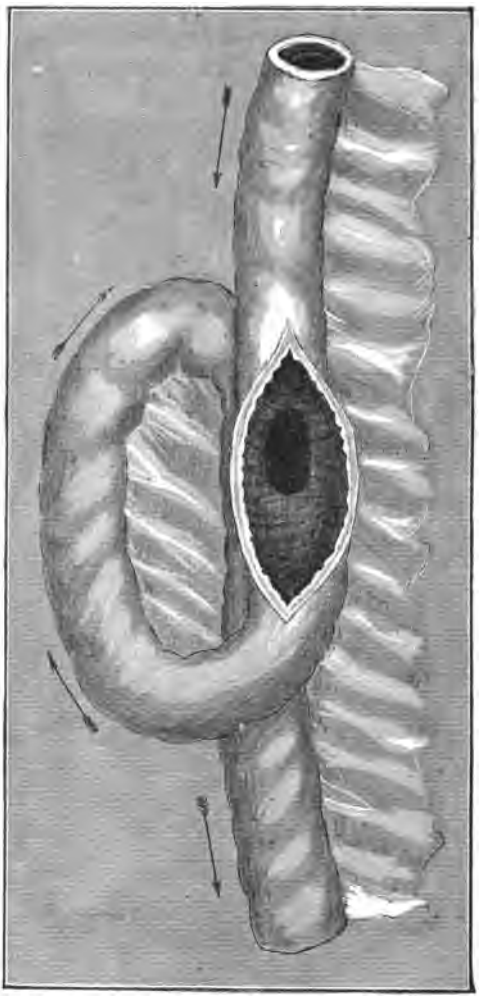


FIG. 44.—A portion of small intestine was short-circuited, an artificial opening between two loops being made by Halsted's method. Specimen obtained at the end of 134 days. The arrows indicate the direction of the intestinal flow. The excluded loop is seen to be diminished in diameter; it was also diminished in length from 20 cm. to 16 cm. The opening was 14 mm. in diameter. ($\times \frac{1}{6}$.)

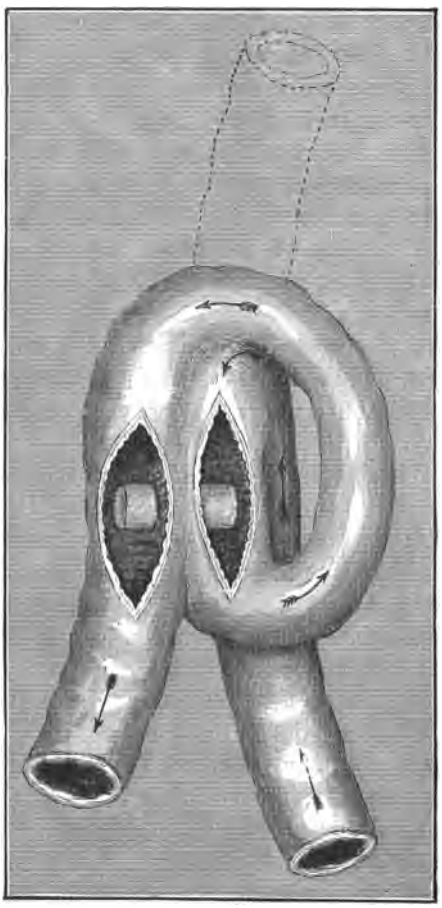


FIG. 45.—Short-circuiting of bowel by Halsted's anastomosis. The arrows indicate the direction of the flow. The upper end is bent by adhesions just before reaching the anastomosis. A style is placed through the artificial opening, which is oval, and measures 8 mm. in its long diameter. The partially excluded partion of bowel is seen to be slightly diminished in diameter. The dotted outline shows the true position of upper end. The opening has much contracted, and this is possibly due to the bend in the upper end not favouring passage through the opening. Specimen obtained after 165 days. ($\times \frac{5}{6}$.)

3. *Changes in the mucous membrane.* On inspection of the figures it will be seen that the mucous membrane of the bowel is different above and below the seat of anastomosis; it is smoother above, the valvulæ conniventes disappear, but apparently only for a time; this change is possibly due to partial obstruction with the consequent pressure and distension; the part of bowel immediately above the junction becomes, in some degree, the large intestine and rectum of the bowel above, and like the large intestine has no valvulæ conniventes.

This explanation, unfortunately, will not agree with our figures of the experiments in which a portion of the bowel was reversed; in these the valvulæ conniventes have disappeared from the normal bowel below the reversed part.

In both these experiments there was a collection of hardened fæces at the upper junction, indicating apparently that, in the upper part of the reversed portion at least, the peristalsis in what has become the reverse direction persisted at the time the animals were killed. Also in Maunsell's operation it will be seen, Figs. 13 and 14, that there is no such marked disappearance of the valvulæ above the seat of union, but that there is some diminution of them opposite the longitudinal incision, which no doubt slightly contracted the bowel.

4. In the case of *implantation* it is to be noted that it makes all the difference whether it is the upper part of bowel that is implanted into the lower or the lower into the upper; in the former case the blind end atrophies, as the peristaltic action tends to empty it; in the latter the peristaltic action in the cul-de-sac is towards its blind end, and consequently this artificial cæcum elongates and its walls become hypertrophied (see Figs. 15 and 16).

In the appendix of man the peristalsis is towards the cæcum; this may explain the smallness of the appendix in man. It will be remembered that in certain pathological conditions which cause obstruction of the appendix, it elongates and its walls become hypertrophied.

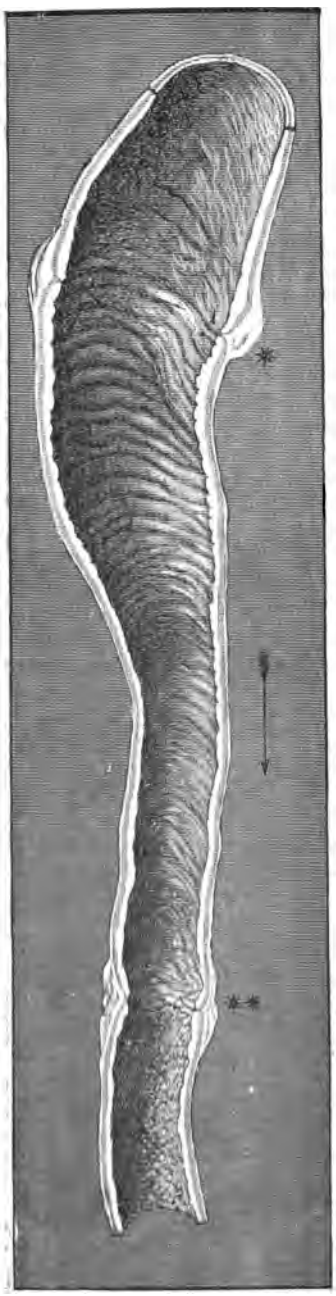


FIG. 46.—Reversal of portion of small intestine, 9½ inches long before division. The reversed portion is now 6 inches long; there was a small collection of fæces at the upper junction, but intestine not distended. Specimen obtained at end of 32 days. (× ⅔.)

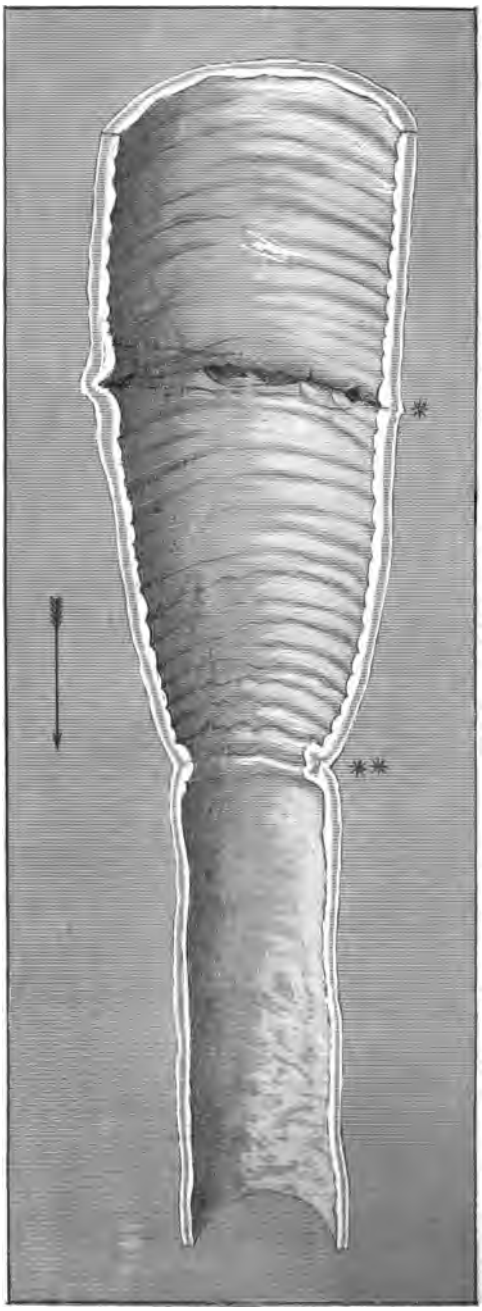


FIG. 47.—67 days. The bowel is distended at upper line of suture, where a mass of hardened fæces was blocked; ulceration along upper line of sutures. The reversed portion has contracted from 5 to 3¼ inches. Lower line of suture perfect. ($\times \frac{2}{3}$.)

5. *Reversal experiments.* In the case of a portion of the intestine being reversed, the reversed portion shortens considerably; in one experiment, at the end of 32 days 9½ inches were reduced to 6 inches (see Fig. 46); in another, at the end of 67 days 5 inches had contracted to 3¼ inches (see Fig. 47).

It is interesting that an animal can live quite well after a length of intestine has been reversed. The difficulty (already referred to) in the upper portion of the reversed part taking up the chyme coming down from above must be due to the nervous mechanism being at fault, continuing possibly the peristalsis in the old way and not adapting itself to the new conditions caused by the operation. This, however, was not sufficient, at least in one of our experiments, to interfere with the enjoyment of good health.

In conclusion, we wish to express our indebtedness to Professor Sherrington, late Professor Superintendent of the Brown Institution, for his suggestions and kind interest in our work.

IV. Abstract of Experiments.

I. Intestinal Anastomosis.

A. *Lateral Anastomosis.*

Exp. 1. *Senn's plates, 63 days.*—Small intestine divided; two cut ends inverted and sutured; intestines incised longitudinally, plates inserted and tied, and continuous suture applied externally. Animal recovered without bad symptoms. Killed 63 days (see Fig. 4).

Exp. 2. *Senn's plates, 68 days.*—Small intestine divided; two cut ends inverted and sutured; intestines incised longitudinally, plates inserted and tied, and Lembert's sutures applied externally. Animal recovered. No bad symptoms. Killed 68 days (see Fig. 5).

Exp. 3. *Mayo Robson's bobbin (made for dogs), 33 days.*—Small intestine, division, inversion and suture of both ends, intestines incised longitudinally, bobbin inserted. Lembert suture externally. Animal recovered, but became thin. Killed 33 days.

Post-mortem.—Small opening. Upper end distended, valvulæ conniventes absent in upper intestine; in lower well marked. No trace of bobbin (see Fig. 6).

Exp. 4. *Davis's catgut plates.*—Experiment attempted and abandoned.

Exp. 5. *Littlewood's bone plates.*—Experiment attempted and abandoned.

Exp. 6. *Murphy's button.*—Leakage and death ensued.

Exp. 7. *Halsted's method of suturing.*—Operation successful; dog did well. Killed 25th day, opening not measured post mortem.

Exp. 8. *Halsted method.*—Operation successful; dog did well. Killed 28th. Opening not measured post mortem.

B.—*End to End Junction.*

Exp. 9. *End to end junction,* 11 *days.*—Small intestine divided, cut ends sewn together by Czerny-Lembert sutures; no bad symptoms. Animal killed 11 days.

Post-mortem.—Lumen more encroached upon than in later specimens (which see). It will also be noticed that here the valvulæ conniventes are equally marked above and below junction, instead of only below as in later specimens (see Fig. 7).

Exp. 10. *End to end suture,* 18 *days.*—Small intestine divided, two cut ends sewn together, mucous membrane to mucous membrane by Czerny sutures, and outer coats to outer coats by Lembert sutures. No bad symptoms. Animal killed 18 days.

Post-mortem.—A slight ridge at line of union; valvulæ conniventes more marked below than above junction (see Fig. 8).

Exp. 11. *End to end suture,* 21 *days.*—Small intestine divided; two cut ends sewn together by Czerny-Lembert sutures. No bad symptoms. Animal killed 21 days.

Post-mortem.—Satisfactory union; slight ridge within bowel; the incurving of outer coats produced by the Lembert sutures is well seen (see Fig. 9).

Exp. 12. *End to end suture,* 35 *days.*—Small intestine divided; two cut ends sewn together; *interrupted sutures in mucous membrane* and *Lembert sutures* in outer coats going down to and through submucous coat (Czerny-Lembert sutures). No bad symptoms. Animal killed 35 days.

Post-mortem.—Satisfactory union. Slight ridge within and supporting tissue without. The incurving of the

coats by the Lembert stitches was well seen (see Fig. 10).

EXP. 13. *End to end suture, 42 days.*—Small intestine divided; two cut ends sewn together end to end; the *mucous membrane by continuous suture,* and one or two additional single sutures; the outer wall of bowel sutured with *Lembert sutures* carried through submucous coat (as recommended by Halsted). No bad symptoms. Animal killed 42 days.

Post-mortem.—Satisfactory union. Slight ridge at line of union (see Fig. 11).

EXP. 14. *End to end suture by Maunsell's method, 30 days.*—Small intestine divided and reunited by Maunsell's method with horsehair stitches. *Four additional Lembert stitches were subsequently applied externally.* The longitudinal incision was made above the junction. It was closed by Lembert's stitches. No bad symptoms. Animal killed 30 days.

Post-mortem.—Good union. The line can be with difficulty recognised, while the longitudinal incision (closed with Lembert's sutures) is shown by a very obvious ridge (see Fig. 13).

EXP. 15. *End to end suture by Maunsell's operation, 78 days.*—Small intestine divided and cut ends united. *The junction produced was by no means perfect, and mucous membrane could be seen externally; therefore several Lembert sutures inserted.* The longitudinal incision also closed by Lembert's sutures. No bad symptoms. Animal killed at end of 78 days.

Post-mortem.—Line of union perfect; considerable ridge at longitudinal incision (see Fig. 14).

EXP. 16. *Jessett's tubes.*—Operation apparently satisfactory, but the dog same day died of another cause.

EXP. 17. *Paul's tube.*—Operation abandoned.

C.—*End to Side Junction.*

Exp. 18. *Implantation of bowel, upper into lower, 84 days.*—Small intestine; intestine divided; upper end of lower bowel inverted and sutured; the end of upper bowel implanted laterally into the side of the lower bowel; Czerny-Lembert sutures. Dog did well and was killed at the end of 84 days.

Post-mortem.—There were some adhesions of the bowel to neighbouring structures; intestine as figured (see Fig. 15).

Exp. 19. *Implantation of bowel, lower into upper, 126 days.*—Small intestine; intestine divided; upper end of bowel inverted and sutured; the end of lower bowel inserted into upper five inches from its inverted end. Dog did well. Killed in 126 days.

Post-mortem.—The anastomosis is free, the blind end being five inches long, the same length as at time of operation; it is also distended, its walls and valvulæ conniventes hypertrophied. Externally sutures in position, the opening not quite full size of bowel (see Fig. 16).

II. Gastro-enterostomy.

Exp. 20. *Gastro-jejunostomy by Halsted's method, 63 days.*—Jejunum found and united, and opened into stomach by Halsted's method, using double-threaded needles which can all be passed in the same and most convenient direction. A portion of the mucous membrane of the stomach was cut away. The cut edges of stomach and jejunum were joined by four additional single sutures. The opening measured 18 × 12 mm. Animal did well, and was killed at the end of 63 days.

Post-mortem.—Firm adhesion between stomach and jejunum; opening measured 10 × 7 mm. Opening between stomach and duodenum not distended: it is not clear how much food passed through the artificial opening. The fact that the opening is smaller now than

at time of operation is explained by the healing of the wound, and there seems no reason to suppose that there would be any further contraction (see Fig. 38).

Exp. 21. *Gastro-jejunostomy, 70 days.*—A gastro-jejunostomy was performed by *Halsted's method;* a linear opening between stomach and jejunum was made, 42 mm. long. Animal did only fairly well; on the 43rd day it was noted that he was well and lively, but extremely thin; on the 70th day he was in better condition; killed that day.

Post-mortem.—A length of jejunum firmly adherent to stomach; intestine between stomach and artificial opening distended; beyond the opening the intestine is rather small; opening between stomach and jejunum measures 18 × 7 mm.; free communication.

Remarks.—It seems probable, owing to the large size of the communication, that some of the food passing along the duodenum re-entered the stomach, and thus circulated round and round; in this way the duodenum would become part of the stomach, and its upper part dilated, the food loitering about there as in the stomach; in this way the existence of the stomach in animals would be due to the presence of a pylorus (see Fig. 39).

Exp. 22. *Gastro-enterostomy by Halsted's method, 76 days.*—A portion of intestine was taken and brought forward, and attached to and opened into the front wall of the stomach by Halsted's method of suture; the dog did well, and was killed at the end of 76 days.

Post-mortem.—It was found to be the ileum which had been attached; the opening was satisfactory, and shows no sign of contraction.

It is noteworthy that in this case, and also in No. 23 experiment, although the route out of the stomach *vid* the pylorus was quite open, yet there was no sign of contraction of the artificial opening, and also that no regurgitation of any consequence seems to have taken place,

for the animals were quite well, and had no vomiting (see Fig. 40).

Exp. 23. *Gastro-jejunostomy, Paul's method, 97 days.* —The abdomen was opened, the jejunum found, and a site selected, an incision made, and the ring inserted and needles passed; the omentum and transverse colon were then turned back, and a site selected on the back of the stomach; on the front of the stomach a vertical incision was made so as not to cut the branches of the gastro-epiploic artery; the needles were then passed at selected site into the interior of stomach and the sutures tied; the portion of stomach included in the ring was *not* divided.

The animal did well, and was killed at the end of 97 days. There was a good-sized opening between stomach and jejunum, showing no signs of contraction. Nothing was seen of the rings. The ivory ring used was kindly sent to us by Mr. Paul (see Fig. 41).

III. Physiological Observations.

Exp. 24. *Short circuiting, 78 days.*—A portion of small intestine was short-circuited by Halsted's method. The length of intestine thus treated was 7 inches (18 cm.). Animal did well, and was killed at the end of 78 days.

Post-mortem.—The short circuited loop is now only 3¼ inches (8 cm.), less than half what it was before. The loop is also diminished in diameter. The position of the loop in the abdomen rather favoured the passage of the chyme through the opening. The artificial opening measured 7 mm. in diameter. When water is injected through intestine, pinching the loop makes no difference in the outflow (see Fig. 43).

Exp. 25. *Short circuiting, 134 days.*—A portion of small intestine is short-circuited, an artificial communication being made by Halsted's method. The length of

short circuited portion was 8 inches (20 cm.). Dog did well, and was quite well when killed at the end of 134 days.

Post-mortem.—Seat of operation readily found. Excluded loop is diminished in length and diameter. Its length is now 6½ inches (16 cm.). The artificial opening is 14 mm. in its longer diameter. On obstructing loop water flows freely through artificial opening (see Fig. 44).

Exp. 26. *Short-circuiting, Halsted's anastomosis,* 165 *days.*—Small intestine short-circuited, opening being made by Halsted's operation; the animal did well, and was killed at the end of 165 days.

Post-mortem.—Short-circuited portion readily found. On injecting water from upper end it flowed partly round the circuit, and partly through the artificial opening. The diameter of the "excluded" part is slightly diminished. The opening measures 8 mm. in its long diameter (see Fig. 45).

Exp. 27. *Reversal of bowel,* 32 *days.*—Small intestine; reversal of intestine; recovery, dog did well. Killed 32nd day.

Post-mortem.—Before division of bowel portion reversed was 9½ inches long, at time of death 6 inches long. Small collection of fæces at upper line of sutures, but intestine not distended: dog quite well when killed (see Fig. 46).

Exp. 28. *Reversal of bowel,* 67 *days.*—A portion of small intestine was divided in two places: the portion thus isolated was, after division, 5 inches long; it was then joined to the intestine again, but in the reverse direction, the lower end being sutured to the upper end, and *vice versâ;* this necessitated a half-turn in the mesentery. The animal did well, bowels acted third day. On the 67th day animal weak and ill, therefore killed.

Post-mortem.—The reversed portion is now 3¼ inches long. A mass of hard fæces at upper junction, causing ulceration of mucous membrane along line of suture (see Fig. 47).

The drawings are by Mr. Lapidge.

V. References.

Ashton and Baldy.—'Medical News,' U.S.A., Feb., 1891.

Barker.—'Lancet,' Jan., 1892, and 'Proc. Roy. Med. and Chir. Soc.,' New Series, vol. ii, p. 257, 1887.

Boyd, Stanley.—'Med.-Chir. Trans.,' vol. lxxvi, 1893.

Davies-Colley.—'Guy's Hospital Reports,' vol. xlviii, 1891.

Greig-Smith.—'Abdominal Surgery,' 5th edit., and private conversations.

Halsted.—'Bulletin of the Johns Hopkins Hospital,' Jan., 1891.

Jessett.—'British Medical Journal,' July, 1889.

Littlewood.—'Lancet,' April, 1892.

Makins.—'Resection of Small Intestine,' 'St. Thomas's Hospital Reports,' 1884.

Maunsell.—'Transactions Intercolonial Med. Congress of Australia,' 1889. (His first operation was in 1886.)

Murphy.—'Medical Record,' (U.S.A.), Dec., 1892.

Paul.—'Liverpool Med.-Chirurgical Journal,' July, 1892.

Robinson.—'Annals of Surgery,' Feb., 1891.

Robson, Mayo.—'Med.-Chir. Trans.,' vol. lxxv, 1892.

Senn.—'Ninth International Medical Congress,' 1887, and 'Intestinal Surgery,' 1889.

Wiggin.—'New York Medical Journal,' Dec., 1895.

ON THE

USE OF GUAIACUM IN THE TREATMENT OF CHRONIC GOUTY AFFECTIONS

AND ITS

VALUE IN WARDING OFF ACUTE ATTACKS

BY

SIR ALFRED B. GARROD, M.D., F.R.S.,

PHYSICIAN EXTRAORDINARY TO HER MAJESTY THE QUEEN; CONSULTING PHYSICIAN TO KING'S COLLEGE HOSPITAL.

Received April 30th—Read May 26th, 1896.

At the meeting of the Royal Medical and Chirurgical Society February 8th, 1848, there was read a paper entitled "Observations on Certain Pathological Conditions of the Blood and Urine in Gout, Rheumatism and Bright's Disease," in which I had the privilege of bringing before this Society several very interesting and important points. In the first place I demonstrated the presence of uric acid in the blood, which had never been proved before although often sought for. Secondly, I showed by many analyses that the uric acid was much more abundant in the blood of gouty patients—that in the blood the uric acid existed in the form of urate of sodium; that in health uric acid is scarcely to be found in the

blood; that in healthy birds, whose urine consists almost entirely of urate of ammonium, uric acid is often altogether absent. These facts were first made known to your Society, and I look upon them as perfectly established by a lengthened experience of forty-eight years. I have mentioned them now as they have reference to matters to which I shall have to allude before I finish this communication.

It can be demonstrated that all truly gouty symptoms are dependent on a morbid condition of the blood, from the presence of a notable quantity of uric acid, or rather urate of sodium, and that deposits of the chalky matter which occur in gouty patients consist chiefly of urate of sodium.

How the uric acid gets into the blood, and where it is formed in the system, we will endeavour to show later on.

I shall first direct your attention to the drug called Guaiacum, which is the resin from the tree called *Guaiacum officinale*, the wood of which is termed Lignum vitæ, also used in medicine on account of the resin contained in it. It can scarcely be said that guaiacum is much used in medicine at the present time; as a powder it is occasionally added to aperients, such as sulphur, magnesia, &c., and in the liquid form it is usually prescribed as the ammoniated tincture. In modern books on Materia Medica the medicinal action of guaiacum is but little commented upon. More was said about it by Cullen and other writers over a hundred years ago, who also spoke of other somewhat allied medicines. Many physicians at that time had an impression that when guaiacum was continuously administered, it possessed some power of warding off gouty attacks, but there seemed to be an idea, very strongly held, that the employment of this drug was by no means free from danger. They looked upon the use of these remedies as suppressing but not eradicating the disease.

At Martinico, more than a century ago, a Mr. Emericon proposed the use of guaiacum dissolved in rum; of this

about a tablespoonful was taken each day, and with very good result, as far as the warding off the attacks was concerned. With regard to the value of this and some other drugs I may quote this statement from Cullen's work: "This fact is certain that from the time of Galen to the present, there are accounts which show the use of certain bitters continued for some time has prevented the return of inflammatory gout."

A rather full discussion on this subject is contained in my work on Gout; but as the readers of this account must be comparatively few, at the risk of a slight repetition I will state my present knowledge of the subject, and how it has been gradually arrived at.

When I first had to treat cases of gout, whether in an hospital or elsewhere, I was accustomed to give salines and alkalines combined with colchicum in moderate doses; under this treatment the patients usually made slow but fairly good progress. After the disease became more chronic in character cinchona was often added to the mixture, and as a rule the improvement continued, and the patient slowly regained his healthy condition. After a time I was induced to try guaiacum, usually as the ammoniated tincture, and I soon became aware of what was gained by this addition; the progress towards recovery was much hastened and the cure in all respects more complete. Perseverance in this plan made the treatment of gouty attacks infinitely more satisfactory, and many patients became so convinced of the action of the Guaiacum that they would not allow themselves to be without it, having it always within reach. I feel sure that I am within the mark when I say that this new method of treatment shortened the duration of the attack to at least one half, allowing the doses of the colchicum to be considerably lessened. It is sometimes desirable to add other remedies to the guaiacum, as iodide of potassium, citrate of lithium, and different salines.

After a short time, meeting with patients who were troubled with attacks of gout at fairly regular intervals,

I was induced to make a trial of guaiacum as a prophylactic.

The first case was a man about sixty-six years of age; for many years he had been accustomed twice a year to have attacks of gout of two or three weeks' duration. He was recommended to continue the diet to which he had been accustomed before he began the treatment, and he was ordered to take early each morning a powder containing twelve grains of guaiacum, the same amount of cinchona bark, a little iodide of potassium and citrate of lithium, with a trace of the corm of colchicum. At first I thought it a necessity to give some colchicum, but the quantity was so insignificant that it could have produced but little or no effect.

For two years and three quarters this patient continued the daily use of the powder, and during the whole of that time he was free from gouty attacks, and never, I believe, had any more during his life. This case was much against Cullen's views, who thought that patients always suffered when their gouty attacks ceased, for his health remained remarkably good after he had ceased to take the powder. When he had passed his eightieth birthday he remarried, and lived till eighty-seven years of age; his death then being hastened by his falling down a flight of stone steps on leaving a dinner party.

Long before this first patient had completed his course, many others were put upon the same treatment. Two of these were men who in the course of their business had to go to China and Japan, and were eighteen months or two years away from England. It had been their lot to be laid up certainly once or twice with attacks of gout in a foreign land. Under this treatment, everything else remaining the same, they went out and returned home again, having been free from any illness during the long journey.

Since the time the above cases were treated so successfully, I have had, I should think, nearly a hundred other patients who have more or less rigorously followed this

treatment, and at the present time many are under the influence of guaiacum. I purposely do not give it alone, because I think much advantage may be obtained from the addition of other remedies. For example, I almost always add citrate of lithium: as I feel sure the beneficial action of the guaiacum is much improved by it, and not infrequently I give the guaiacum as a cachet, followed by a very pleasant draught of the effervescing citrate. Neither can be regarded as medicines, seeing they may be taken indefinitely in the same way as food.

Before concluding this account of the action of guaiacum, I may here relate a circumstance which has occurred to me many times. Patients to whom I have prescribed guaiacum after taking it for a few days have come again complaining of the effect which the guaiacum has had on the urine; that whereas before taking it the urine was very clear and healthy-looking, since its administration it had become thick and looking very unhealthy; when, however, these same patients were asked about their health, they all acknowledged they were feeling better, and in all respects going on satisfactorily. Without at present endeavouring to explain the action of guaiacum upon the urinary secretion, it is quite certain that it frequently causes the copious elimination of uric acid in the form of urates, which probably is in close relation to its efficacy in gouty cases.

I often give the guaiacate of lithium in the form of pills. It is a preparation I introduced at least twenty years ago, and which seems to act very efficiently.

Cullen and others, who recognised the effect of guaiacum in arresting gouty attacks, considered that the same power resided, at any rate in some degree, in other medicinal substances, and practical experience has taught me that Serpentary, the rhizome and rootlets of *Aristolochia serpentaria*, is one of these substances. When from any cause a patient is unable to take guaiacum for any length of time, a circumstance which seldom occurs, then serpentary seems to be able to take its place; and

I have several patients, usually elderly men, who have found great benefit from this medicine in their gouty attacks, and several who have been most unwilling to be ever without it, but as yet I can give no such evidence of its prophylactic powers as I can in the case of guaiacum.

I give the serpentary in the form of extract as well as in that of a tincture.

Before concluding my remarks on the action of drugs on gouty conditions of the system, it is of much importance that colchicum should be alluded to.

Every one who has had any experience in the treatment of gouty cases must be aware that this drug possesses very great power of controlling the inflammatory action which occurs in the articular structures, and that in the treatment of attacks of gout it is perhaps unequalled, but as a prophylactic its value is more doubtful, and few would like to give it in doses sufficiently large to keep the disease at bay for any very lengthened period. I believe that colchicum possesses the greatest power of checking gouty inflammation when present, but little or none in preventing its recurrence.

There is also a feeling in the minds of many of the public, also of some of the profession, that colchicum although curative of existing gouty inflammation, possesses also the power of causing its rapid recurrence, and that in cases of gout treated by efficient doses of this drug there is a tendency to relapse. I must confess that in my own experience I have never noticed this, except in cases treated by quack medicines containing large quantities of colchicum, when the relapses have been frequent and very unyielding.

I think we may safely say that although colchicum is a most potent and valuable remedy in gouty inflammation, it is not one that can be given for a very lengthened period of time in large doses, and that it is far inferior to guaiacum as a prophylactic.

In order to attempt to give an explanation of the action of guaiacum in relieving and keeping off attacks

of gout, it will be necessary to state my views of the formation and secretion of uric acid in the animal body. Some fifty or sixty years ago it was considered that uric acid was produced by the kidney itself; it had been sought for in the blood, but without success; when, however, this principle was found in the circulating fluid, the ideas of its origin in the body were changed, and up to the present time the view held by physiologists is as follows :—in the metabolism of the nitrogenised tissues of the body, urate of ammonium is first formed, which in some animals is at once thrown out by the kidney; for instance, this is the case in all birds and in most reptiles; in other animals a further change takes place—urea is formed from the urate of ammonium and in that condition eliminated by the kidneys.

When carefully examined the objections to this hypothesis are very numerous and to my mind insuperable. They will be found detailed in a paper in the Proceedings of the Royal Society, vol. lviii. In the same communication I stated my own views derived from a thorough investigation of the facts. The opinions I hold on the subject amount to this :

1. That in the metabolism of the nitrogenised tissues of the body urea is first formed. I found urea in the blood of birds whose urine usually contains none of this principle, as well as in the blood of mammals whose urine contains it in abundance.

2. That uric acid in the form of urate of ammonium is formed in the kidneys themselves from urea (not urea from urate of ammonium) and is thrown out in that form.

3. When uric acid (as urate of sodium) is found in the blood I consider that this arises from the absorption from the kidney structure, where in man it is apt to be retained for a time; but in the blood of the healthy bird, such as the pigeon, whose excretion is entirely urate of ammonium, no uric acid can be detected; this was stated in my communication to this Society in February, 1848.

Holding this view of the origin of uric acid, we can

explain the action of guaiacum in this way. We must assume that guaiacum acts as a stimulant on the kidney itself, and causes that organ to throw off any urate of ammonium remaining in its structure, thereby preventing absorption into the blood. If this is the case, it can be easily understood why its continued use prevents any accumulation from taking place, and prevents the blood from being contaminated by its absorption. If this view is correct it is worth while to investigate the action of guaiacum in cases where calculi are being formed.

With regard to the action of guaiacum, it may be confidently asserted that it has no chemical action on uric acid or urate of ammonium, so no clue to its action can be obtained from its chemical action; it has no solvent power of any kind, its action appears to be a stimulant on the capillary blood vessels, not on the heart itself, and it is on the mucous membranes that its action is most evident, especially on the genito-urinary organs.

In conclusion, I hope I have been successful in establishing the following points as to the action of guaiacum:—

1. Guaiacum is an absolutely innocuous substance, and may be taken for an indefinite period of time; it may be looked upon rather as a condiment than as a drug; it is as innocent as ginger or any other spice.

2. Guaiacum possesses a considerable power, less than colchicum, in directly relieving patients suffering from gouty inflammation of any part; it may be given whenever the patient is not very feverish.

3. Guaiacum taken in the intervals of gouty attacks has a very considerable power of averting their recurrence, even over a lengthened period; it is, in fact, a very powerful prophylactic.

4. Guaiacum from long-continued use does not lose its prophylactic power.

5. Guaiacum, although it can be taken by most individuals, is apt in a few cases to cause irritation of the bowel, so as to prevent its administration as a remedy in gout. In such cases serpentary may be often substi-

tuted with advantage, although it is as yet doubtful whether it equals guaiacum in its prophylactic power.

If the statements contained in this communication have the effect of inducing my medical brethren to direct their attention to the value of guaiacum in the treatment and prevention of gout, I feel I shall have conferred some benefit on the profession, and through them on the public at large.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 230.)

CASE OF SPLENIC ANÆMIA

(SPLÉNO-MÉGALIE PRIMITIVE)

WITH AN ACCOUNT OF THE DISEASE

BY

SAMUEL WEST, M.D.

Received February 9th—Read June 9th, 1896.

WALTER D—, a horsekeeper, æt. 36, came to see me in the out-patient room at St. Bartholomew's Hospital, complaining of losing flesh and feeling very ill. He looked extremely feeble, was sallow and pallid, had a raised temperature, and a very large spleen. I thought the case might be one of leucocythæmia, but the hurried examination of the blood in the out-patient room did not reveal any increase of white blood cells. The patient was then admitted into the hospital.

The patient gave the following history:—He was quite well until one year ago, when he was troubled with a severe cold and pain across the loins. In January, 1894, a tooth was extracted, and this was followed by profuse bleeding. The patient said he lost about a quart of blood, bleeding continually from 7.30 p.m. till noon of the following day. He was in bed for about a week after this hæmorrhage. From that time till now he had been getting weaker and weaker and losing flesh. When in health he weighed fifteen stone, and now weighs not much more than nine.

Shortly before the extraction of the tooth he began to suffer with bleeding from the nose. This occurred chiefly during attacks of coughing, and from that time till now his nose has bled occasionally, and he has been spitting blood from time to time, which comes, he thinks, from the nose. His wife says he has been getting weaker and thinner for the last twelve months.

There is nothing in the previous history of the patient, nor in the family history, which has any reference to the case.

The patient has been a temperate man, and has never had syphilis.

The patient looks sallow, cachectic, and very ill. He is extremely pale, but the cheeks are flushed. His temperature, on admission at 7 p.m., was 103°. The patient is constantly spitting some blood-stained fluid which looks like saliva, the fauces are congested, and the gums a little vascular. The bleeding probably comes from the nose or back of the pharynx.

An examination of the respiratory organs revealed nothing abnormal, but the voice was hoarse, and on laryngoscopic examination the cords were seen to be reddened, with a little blood on the upper surface, but nothing else abnormal was found. The cardiac dulness was slightly increased, and the heart somewhat dilated, but there was no other sign of disease. A blowing systolic murmur was heard all over the præcordium, especially over the pulmonary area. There was somewhat increased pulsation in the vessels of the neck. The pulse was 96, of normal tension and fair volume, the artery being a little thickened.

Examination of the abdomen showed that the liver was somewhat enlarged, the upper level being in its normal position, but the lower an inch or an inch and a half below the edge of the ribs in the right nipple line. On the left side of the abdomen was a hard tumour, extending downward in the direction of the right pubes, and reaching to the umbilicus in the middle line and to a point about one inch from the anterior superior spine of the ilium on the left side. It was continuous with the splenic dulness, which extended four inches or more above the costal arch. The tumour was not tender nor painful, it moved freely on respiration, was dull to percussion, smooth on the surface, and in its centre towards the middle line a deep notch was to be felt. It was evidently the spleen.

The urine was normal, 1020 specific gravity, containing no albumen and no blood. There was no tenderness of the bones, nor any defect in the neuro-muscular system. The left pupil was a little larger than the right; ophthalmoscopic examination showed the retinæ to be quite healthy although somewhat pale.

The extreme anæmia and illness, associated with the large spleen, suggested leucocythæmia, and the blood was examined from this point of

view, and although the white blood cells were somewhat increased, they were not sufficiently increased to justify that diagnosis. The following estimate was made; red corpuscles, 2,055,000; white corpuscles, 50,000; hæmoglobin, 25 per cent. of the normal.

The patient was put upon arsenic and quinine, and a chlorate of potash gargle was given him for the throat and mouth.

On December 3rd, a week later, the following note was taken:—The patient's general condition has not improved, the temperature remains high, reaching on some days 103°. He has had some trouble with sleep, but a little paraldehyde has been sufficient to give him rest, and he can now sleep without it. He complains of cutting pain across the abdomen, not specially localised. His hoarseness has increased, but there is nothing special to be observed in the larynx. The urine is rather high-coloured, but otherwise not abnormal. The bleeding from the gums has practically ceased, though the sputum is now and then somewhat tinged with blood. The gums are less vascular.

The chest was carefully re-examined and nothing fresh found, but the pulsation in the carotids seemed to be somewhat more marked, and the pulse was now of low tension. A few crepitations were heard at the base of the lungs, probably due to position.

The arsenic had been gradually increased, so that now the patient was taking about 10 minims three times a day. This did not in any way disturb the digestion.

December 14th.—The general condition of the patient had considerably improved during the last few days, his voice was less hoarse, but he had begun again to split blood or blood-stained saliva, and was now spitting up as much as when he was admitted. The temperature had been lower during the last few days by nearly a couple of degrees on the average. It was still markedly hectic, falling considerably towards morning and rising to its maximum in the evening. Another examination of the blood was made with the following result: red cells, 2,226,000; white cells, 75,000; hæmoglobin, 35 per cent. of the normal. The proportion of red cells to white was as 29 to 1. There were also many masses of blood platelets, resembling in colour the red blood cells though much smaller.

On the 17th another examination of the blood was made: red cells, 1,900,000. Leucocytes 58,300, stained deeply with Toison's fluid. The maximum temperature last night was 103°.

On the 18th the patient had some more bleeding from the nose, which came evidently from the right nostril. No bleeding point could be seen either on the turbinate bone or on the septum when the nose was examined with the speculum. The nostrils were syringed with cold water, and then with Liq. Ferri Perchlor., after which the bleeding stopped.

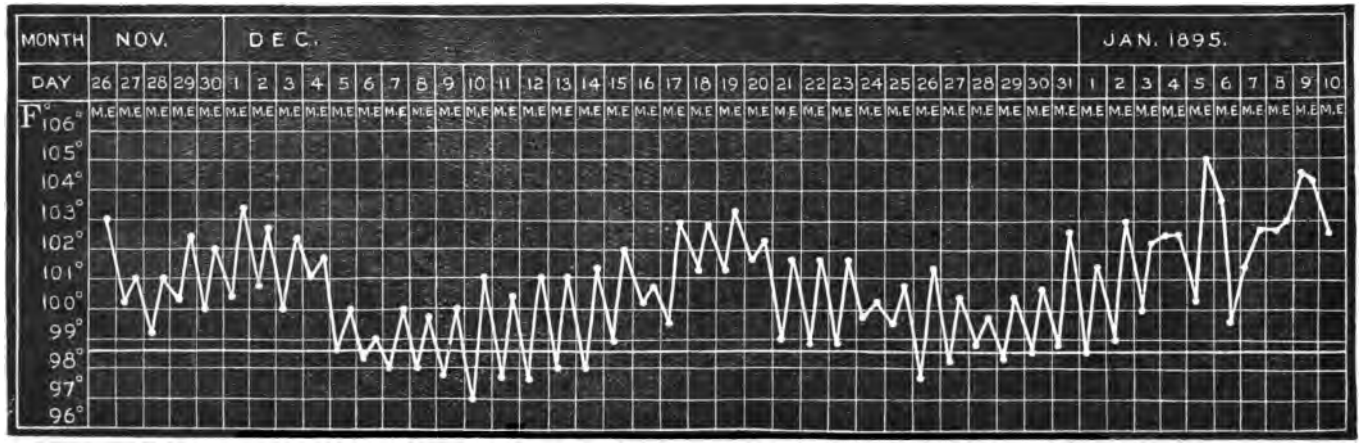
On December 27th the eyes were examined again, and in the left fundus oculi a very large hæmorrhage was found just outside the disc. The patient had lately become very hoarse. His general condition had in no way improved, he was extremely pale and ill, and was obviously losing ground. The fever remained much as before.

On January 3rd, as the hoarseness still continued and seemed to get worse, the larynx was examined, and the arytænoids were found somewhat swollen; two days later, on the 5th, the patient's breathing became very laboured, with marked inspiratory stridor and some recession of the soft parts of the chest. The patient was placed in a steam tent, but the stridor and recession increased until the dyspnœa became so extreme that at six o'clock on the morning of the 5th tracheotomy was performed under chloroform. The operation gave considerable but not complete relief, though towards evening the breathing became much less difficult, and the patient more comfortable. Later, however, the temperature reached 105°, and the patient became much worse.

The examination of the larynx made shortly before the operation showed that the patient had an acute swelling of all the parts about the larynx, viz. the arytænoid cartilages on both sides, the epiglottis, and the epiglottic folds, as well as the false chords. The condition was regarded as one of acute œdema of the glottis. After the operation the œdema greatly subsided, but the patient was not able to do without the tube. The tracheotomy wound was very deep, and took on an unhealthy character from the first, becoming foul and sloughing, especially in its deeper parts. There was also at times a good deal of venous bleeding from the wound. On the 8th the tube was left out and remained out all night, the wound in the meanwhile being cleansed as much as possible, and fomentations applied. The patient was able to breathe fairly through the mouth, and could speak without much hoarseness, but it was thought right the next day to replace the tube, and the parts around were packed closely with gauze on account of hæmorrhage, the exact source of which could not be ascertained, and it seemed to be a general oozing from the surface. There was no hæmorrhage, however, on that night, and the patient slept well after an injection of morphia.

On the afternoon of the 10th there was a sudden return of bleeding, the patient coughed violently, and expelled the plugs round the tube. He then seemed to suck blood into his trachea, and died suddenly.

The patient had been treated with arsenic throughout, the dose being gradually increased until it reached 12 minims of Liquor Arsenicalis three times a day; this was combined with iron. Ergot was also administered from time to time with the object of controlling the hæmorrhage, but without much result. The diet consisted of such light food as the patient was able to take, and he had stimulants to the extent of four or five oz.



Temperature chart giving the maximum (evening) and minimum (morning) temperature daily.

daily of brandy, though just at the last, on account of his extreme feebleness, the amount was increased to 8 oz.

The reports of the blood which have been given in the preceding report were made in the wards by Dr. Herringham and Mr. Lloyd. Dr. Kanthack was asked to examine the case in the laboratory, but his account differs from the preceding report. On January 4th, 1895, he reported that there was no leucocytosis; and of the few leucocytes present most were lymphocytes.

A post-mortem examination was made of the body twenty-four hours after death. The heart weighed 12 oz. All its cavities contained post-mortem clots. The endocardium of the left ventricle was thickened, the mitral valves competent, but the aortic valves slightly incompetent, a vegetation existing on one of them as large as a pea. The lungs were œdematous and somewhat emphysematous, but otherwise normal. The larynx was still in a condition of œdema, and there was a slight superficial ulceration of the vocal cords. The kidneys weighed 21 oz., and were not obviously abnormal. The liver weighed 93 oz.; it looked somewhat granular, suggesting an early condition of cirrhosis; no iron reaction was obtained. The spleen weighed 76 oz.; it was very soft and contained one small infarct; though so large, it did not present any abnormal appearance.

Portions of the different organs were retained and sent to the pathological laboratory for full report. The following account of the specimens has been given me by Dr. Kanthack.

The liver and spleen were hardened in a mixture of glycerine and spirit, and the heart in Müller's fluid.

Spleen.—The spleen shows slight increase in the trabecular tissue, *i.e.* slight fibrosis. The Malpighian bodies are much diminished in size and badly developed. The cellular elements are almost entirely lymphocytes. Only an occasional large cell with several nuclei, hardly worthy of the name giant-cell, can be detected. Iron reaction negative, both macroscopic and microscopic.

Liver.—Iron reaction negative, both macroscopically and microscopically. Liver-cells arranged in trabeculæ which are separated by an intercellular substance. This substance in many parts consists of connective tissue, in other parts there are round-cells. In many places red blood-corpuscles are found between the liver-cells. Where these collections of red blood-corpuscles are most evident, the liver-cells have atrophied. The liver presents thus: (1) intercellular or cirrhotic changes; (2) numerous capillary hæmorrhages; (3) cellular infiltration.

Heart.—There was no fat detected in the heart muscle.

Splenic Anæmia.

There is a form of profound anæmia, progressive in character, generally of no long duration, associated with great enlargement of the spleen, but without leucocytosis or enlarged glands. The disease is very rare and peculiar in its character, and therefore seems to deserve a special name.

Splenic Anæmia seems to be the name under which most cases are described in this country, but the disease has also been called *Splenic Cachexia, Splenic Pseudo-leukæmia, Lymphadenoma Splenica,* as well as *Splenomegalie Primitive,* under which name it is described chiefly in French literature.

Bruhl, though not the first to describe the disease, has given us the best account of it, and it is from this that the following description is chiefly derived.

There are three stages in the disease: the first the preliminary stage, in which there are no other symptoms except those of gradually increasing weakness, with perhaps occasional attacks of pain in the splenic region; the second where the anæmia becomes marked and the spleen greatly enlarged; and the final stage that of cachexia and death from asthenia.

The *initial symptoms* of the disease are those of great asthenia, similar to that which occurs in Addison's disease, and progressive anæmia. There are, at the same time, a good deal of wasting of muscle and loss of muscular power, usually without emaciation, the panniculus adiposus being well maintained.

Appetite and digestion are often preserved for some time, and other functions undisturbed. All the other symptoms in this stage are merely those common to all forms of profound anæmia, viz. shortness of breath, pain in the cardiac region, palpitation, and occasionally some swelling of the feet. There is at this stage nothing characteristic in the symptoms.

In the *second stage*, attacks of severe pain are complained of in the left hypochondriac region, with tenderness over the spleen, the pain radiating to the back, left shoulder, and especially to the lumbar region. It is intense enough to resemble colic, and except that it is felt on the left side, is very like hepatic-colic in character. There is generally a history to be obtained at this time of increasing pallor, loss of strength, and of attacks of epistaxis for some time previously. The temperature is now found to be raised, varying from 102° to 104°, and at the same time the digestive system is greatly disturbed. It is in this stage that most of the patients come under observation, or rather it is in this stage that the disease is first recognised.

The spleen is then found to be considerably enlarged, and tender; though usually smooth, it may be somewhat irregular on the surface. Signs of local peritonitis in the splenic region may be present, or those of left basic pleurisy,—in both cases due to an inflammation spreading from the spleen; and sometimes there may be found some general peritoneal effusion.

The heart may be dilated, and blowing murmurs may sometimes be heard, as in other forms of anæmia.

The blood shows no characteristic pathological changes; the condition is simply that which is met with in all extreme anæmias of simple origin. The red cells are diminished to one half, one fourth, or less; their form is preserved, but they are a little reduced in size. There is no poikilocytosis, but the cells are poor in hæmoglobin, this being reduced to one half or one sixth, and this loss is in excess of the diminution in the number of blood cells. Occasionally there is a slight increase in the white cells, but not more than the fever or some intercurrent malady would account for, and there is nothing at all similar to what is found in leucocythæmia. No micro-organisms have been discovered on microscopical examination or by cultivation.

The spleen is observed gradually to increase in size,

but occasionally between the attacks it seems to become for the time smaller. The recurrent attacks of pain in the splenic region, apparently due to perisplenitis, often cause very great suffering.

The liver is also often somewhat enlarged, and is sometimes associated with slight jaundice.

The digestion is considerably disturbed, and the appetite is lost; there is a good deal of nausea, and sometimes obstinate vomiting, which occurs in such paroxysmal attacks as almost to constitute crises, and they often coincide with attacks of abdominal pain. There is usually troublesome constipation, but occasionally diarrhœa, almost dysenteric in character, with tenesmus and discharge of bloody mucus.

Hæmorrhages are common, usually in slight degree, and of the nature of oozing; this may be difficult to control and add greatly to the anæmia. Profuse hæmorrhage from any part is uncommon.

Epistaxis is very frequent, and is often one of the earliest symptoms, and usually slight; it is of importance chiefly on account of its frequent occurrence. It is sometimes severe, and may even require plugging of the nostril.

Oozing from the gums, again, is not uncommon, and is very difficult to control.

From the gastro-intestinal organs, hæmorrhage is rare, and if in any large amount, is probably associated with some secondary lesion; a case is recorded by Douglas Stanley, in which profuse and fatal hæmatemesis took place, and no ulceration of the stomach was found; but in other cases gastric ulcers have been described, and Williamson records a case of fatal hæmorrhage from the bowels, in which an ulcer was found in the small intestine.

Hæmaturia and hæmoptysis are also met with, but these are rarer than other forms of hæmorrhage.

In the skin small petechiæ on the lower limbs are common, especially in patients who are not in bed, but they are of no special significance. A purpuric eruption of greater degree than this is not described.

Urinary changes are indefinite, and vary much, but sometimes there is albumen present.

No doubt hæmorrhages may occur into the retina, as they do in other forms of anæmia, but I do not know that they are recorded except in the case I have described.

Bruhl states that fever is unusual, but in some of the cases recorded the temperature reached a considerable height, and was of the nature of an irregular hectic, rising to 103° or 104° every evening.

The last stage of the disease is characterised by progressive asthenia ending in death, but there is nothing characteristic nor different from what is usually met with in cases of profound anæmia, except that the hæmophilic symptoms may be unusually pronounced.

The course of the disease is continuously progressive, though there appear to be periods of temporary arrest.

The duration of the disease is short, from six months to two years, but it is occasionally more prolonged; thus, Müller records one of which the duration was four years and a half.

It is possible that cases may be of even shorter duration than six months; thus Erbstein records one of this kind under the name of Pseudoleucæmia Acuta.

Complications may hasten the end; for example, pneumonia, bronchitis, abscess or peritonitis in connection with the spleen, and as described, sometimes severe hæmorrhage from the stomach or intestines, possibly connected with an ulcer there. In some cases death is sudden, from cardiac failure.

The diagnosis is as a rule easy, though the cases themselves are very rare. The diagnosis has to be made from leucocythæmia, from Hodgkin's disease, from other grave anæmias, from the cachexia of malignant disease, from pernicious anæmia, and lastly, from other forms of splenic

enlargement. In all other pernicious anæmias except this, splenic enlargement is very rare.

The ætiology of the disease.—The affection may occur at any age, but it is commonest in the adult, and most frequent by far in males, only two or three cases being recorded at all in the female.

The causes are unknown, but the ordinary causes of splenic enlargement are absent.

Morbid anatomy.—The spleen may be twelve inches long, and weigh from two to seven pounds. It is firm, with notches which are well marked, and there are occasionally irregularities on the surface. It is reddish-brown in colour, and it is often surrounded by a thickened capsule, which may be adherent either to the diaphragm or to the parts near. On section it looks as if there were a general hypertrophy of the organ, but occasionally there are hæmorrhagic infarcts in it. The surface of the section is dry and yields little juice, and it shows greyish streaks or fine granulations. The former prove on microscopical examination to be thickened trabeculæ, the latter fibrotic Malpighian corpuscles. The central artery is much thickened, and occasionally the whole corpuscle seems to have undergone complete sclerosis and atrophy. With the thickening of the trabeculæ there has been great disappearance of the spleen-cells and dilatation of the veins.

The characteristic changes are: (1) fibrosis of the organ; (2) the disappearance of the pulp; and (3) especially the cirrhosis and atrophy of the Malpighian bodies.

In leucocythæmia the Malpighian bodies as a rule are hypertrophied, that is to say, the condition of the spleen is one of hypertrophy; on the other hand, in this disease the condition is one of atrophy and has been called cirrhosis.

The liver is slightly cirrhosed, with much pigmentation. The cells are misshapen, atrophied, and often granular. But, as far as one knows, there is no iron reaction.

The pancreas also is occasionally indurated, it is stated, but this is by no means constant.

The lymphatic glands are normal.

The bone marrow is also usually normal, but in one or two cases it is described as red and infiltrated with leucocytes.

There are no definite changes in any other organ besides those which occur as the result of anæmia.

Bruhl argues in favour of the splenic change being primary and the cause of the disease, attributing the cachexia to the loss of function of the spleen and its interference with the chemical composition of the blood.

The treatment must be systematic, and does not differ from that of other anæmias, but so far nothing has been found to check the progress of the disease.

The following points of interest deserve attention in the case which I have described. (1) The acute onset and short duration of the disease, for the patient believed himself to be well until one year before his death. (2) The hæmophilic condition which developed so early, for epistaxis was one of the first symptoms, and profuse bleeding after the extraction of a tooth occurred soon after; later, the bleeding from the gums was troublesome, while in the last few weeks of life there was frequent hæmoptysis, the blood coming probably from the trachea, and possibly the larger air tubes; and at last came the hæmorrhage into the retina, and also from the tracheotomy wound. (3) The œdematous condition of the larynx and the necessity for tracheotomy, which operation, with the bleeding which took place from the tracheotomy wound, was no doubt the immediate cause of death. (4) The elevation of temperature, which was continuous as long as long as the patient was under observation.

The number of cases hitherto recorded is small, for in 1891 Bruhl could only collect fourteen cases, and two of

these only were in the female sex. Since then a few others have been published, but the total number is probably still under twenty.

The claim that these cases form a definite clinical group of anæmia must, I think, be regarded as fully established.

They appear to me to resemble most closely some cases of severe malignant ague, and if this be so it would suggest that a blood parasite was probably the cause of the disease, but careful examinations of the blood have hitherto proved negative.

On the other hand, it may be true, as Bruhl has suggested, that the symptoms are to be referred primarily to the changes in the spleen, and to perverted nutrition caused by the atrophy of the organ. Still, the acuteness of the symptoms, and the rapidity with which the anæmia develops, seems to be somewhat inconsistent with its reference to a chronic disease like cirrhosis.

BIBLIOGRAPHY.

Bruhl.—Arch. gén. de Méd., 1891, i and ii.
„ Gaz. des Hôpitaux, 1891.
Gobain.—Semaine Médicale, 1887.
Banti.—Dell' Anæmia Splenica, 1882.
Pel.—Berl. klin. Woch., 1885 and 1887.
Renvers.—Deut. med. Woch., 1888.
Ebstein.—Deut. Arch. f. klin. Med., xliii.
Luzet.—Thèse de Paris, 1891.
Keating.—Dis. of Children, vol. iii (Griffith).
Carr.—Lancet, April 23rd and 30th, 1892.
Williamson.—Medical Chronicle, May, 1893.
Douglas Stanley.—Brit. Med. Journ., 1895, ii, 1298.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. viii, p. 263.)

INDEX.

The Indices to the annual volumes are made on the same principle as, and are in continuation of, the General Index to the first fifty-three volumes of the 'Transactions.' They are inserted, as soon as printed, in the Library copy, where the entire Index to the current date may always be consulted.

ABSCESS: Prevesical abscess (Ernst Michels, M.D.) . 63

ANÆMIA: Splenic anæmia (spléno-mégalie primitive) (Samuel West, M.D.) 823

ANÆSTHETICS: Posture in its relation to surgical operations under anæsthetics (Frederic Hewitt, M.D., and A. Marmaduke Sheild) 1

ANASTOMOSIS: Observations and experiments on intestinal and gastro-intestinal anastomosis (Walter Edmunds and Charles A. Ballance) 255

Andrews, Launcelot, M.B.: obituary notice . . . ciii

BALLANCE, Charles A.

A case of neoplasm cyst of the brain treated by operation 167

——— and *EDMUNDS, Walter.*

Observations and experiments on intestinal and gastro-intestinal anastomosis 255

BALNEOLOGY: see *Climatology.*

BLADDER: Prevesical abscess (Ernst Michels, M.D.) . 63

Bostock, John Ashton, C.B.: obituary notice . . . ciii

BRAIN: Case of neoplasm cyst of brain treated by operation (Charles A. Ballance) 167

BRONCHIAL TUBES: on the effects produced by the retention of foreign bodies for lengthened periods in the bronchial tubes (Rickman John Godlee) 197

Bruce (Dr. J. Mitchell, *Hon. Secretary*) . . . cxiii

Buchanan, Sir George: obituary notice . . . cix

CLIMATOLOGY and BALNEOLOGY: Report of Committee on Climatology and Balneology of Great Britain . lxxxv

——— ——— publication and presentation to the Society of first volume of the Report . . . lxxxv

COLIC (RENAL) in infants (R. A. Gibbons, M.D.) . 41

COLLES' LAW: The exceptions to Colles' law (George Ogilvie, M.B.) 87

COUNCIL: Report of the Council . . lxxxiii

Crawford, Sir Thomas: obituary notice . . ciii

CYST: Neoplasm cyst of brain treated by operation (Charles A. Ballance) 167

DEBENTURE LOAN: Payment . . lxxxviii

Dickinson, William Howship, M.D. (*elected President*) . xci

Dixon, James, obituary notice . . civ, cv

Durham, Arthur Edward: obituary notice . . cviii

ECCHYMOSES from natural causes (H. A. Lediard, M.D.) 75

EDMUNDS, Walter, and *BALLANCE, Charles A.*
Observations and experiments on intestinal and gastro-intestinal anastomosis . . . 255

ENDEMIC FEVER of the Mediterranean (Surg.-Capt. M. Louis Hughes) 209

FEVER (malarial): see *Malaria.*

— see *Endemic Fever* of the Mediterranean.

FOREIGN BODIES: On the effects produced by the retention of foreign bodies for lengthened periods in the bronchial tubes (Rickman John Godlee) . . 197

GARROD, Sir Alfred B., M.D., F.R.S.
On the use of guaiacum in the treatment of chronic gouty affections, and its value in warding off acute attacks 313

GASTRO-INTESTINAL and intestinal anastomosis (Walter Edmunds and Charles A. Ballance) . . 255

GIBBONS, R. A., M.D.
Renal colic in infants 41

GODLEE, Rickman John.
On the effects produced by the retention of foreign bodies for lengthened periods in the bronchial tubes . 197

GOUT: Use of guaiacum in the treatment of chronic gouty affections, and its value in warding off acute attacks (Sir Alfred B. Garrod, M.D., F.R.S.) . . 313

GUAIACUM: Use of guaiacum in the treatment of chronic gouty affections, and its value in warding off acute attacks (Sir Alfred B. Garrod, M.D., F.R.S.) . . 313

HEWITT, Frederic, M.D., and *SHEILD, A. Marmaduke.*
On posture in its relation to surgical operations under anæsthetics 1

HUGHES, Surg.-Capt. M. Louis.
Note on the endemic fever of the Mediterranean. Communicated by Dr. George Thin . . 209

HUTCHINSON, Jonathan, F.R.S.
Address as President at Annual Meeting . . xcvii

Huxley, Thomas Henry: obituary notice . . cx

INTESTINE: Intestinal and gastro-intestinal anastomosis (Walter Edmunds and Charles A. Ballance) . 255

KIDNEYS: renal colic in children (R. A. Gibbons, M.D.) 41

Lane, Hugh: obituary notice . . . ciii

Larrey, Baron H.: obituary notice . . . ciii

LEDIARD, H. A., M.D.
Ecchymoses from natural causes . . . 75

LIBRARIANS: Report of Honorary Librarians lxxxv

LIVER: Excision of portion of liver for tumour (Mayo Robson) 159

Longmore, Sir Thomas: obituary notice . . ciii

MacAlister (J. Y. W., *Resident Librarian*) . lxxxvii, xc

MALARIA: The parasite of malaria as observed in the malarial fevers of the south of Spain (Robert J. Marshall and George Thin, M.D.) 101

— (PERNICIOUS): Note on the appearances found in the tissues in a fatal case of pernicious malaria at Sierra Leone (George Thin, M.D.) . . . 143

MARSHALL, Robert J., and *THIN, George, M.D.*
The parasite of malaria as observed in the malarial fevers of the south of Spain . . . 101

MEDITERRANEAN: Endemic fever of the Mediterranean (Surg.-Capt. M. Louis Hughes) . . 209

MICHELS, Ernst, M.D.
On prevesical abscess 63

NATURAL CAUSES producing ecchymoses (H. A. Lediard, M.D.) 75

NEOPLASM CYST OF BRAIN treated by operation (Charles A. Ballance) 167

Obituary Notices of deceased Fellows, 1895–6.

Andrews, Launcelot, M.B. . ciii	Huxley, Thomas Henry . . cx
Bostock, John Ashton, C.B. ciii	Lane, Hugh ciii
Buchanan, Sir George . cix	Larrey, Baron H. . . . ciii
Crawford, Sir Thomas . ciii	Longmore, Sir Thomas . . ciii
Dixon, James . . civ, cv	Pasteur, Louis cxi
Durham, Arthur Edward . cviii	Savory, Sir William, Bart. civ, cvi

OGILVIE, George, M.B.
The exceptions to Colles' law . . . 87

PARASITE OF MALARIA as observed in the malarial fevers of the south of Spain (Robert J. Marshall and George Thin, M.D.) 101

Parker (R. W., *Hon. Secretary*) . . . cxiii

Pasteur, Louis: obituary notice . . . cxi

POSTURE in its relation to surgical operations under anæsthetics (Frederic Hewitt, M.D., and A. Marmaduke Sheild) 1

PREVESICAL ABSCESS (Ernst Michels, M.D.) . 63

RENAL COLIC in infants (R. A. Gibbons, M.D.) . 41

ROBSON, A. W. Mayo.
Excision of a portion of liver for tumour . . 159

Savory, Sir William, Bart.: obituary notice . civ, cvi

SHEILD, A. Marmaduke, and *HEWITT, Frederic, M.D.*
On posture in its relation to surgical operations under anæsthetics 1

SIERRA LEONE: Note on the appearances found in the tissues in a fatal case of pernicious malaria at Sierra Leone (George Thin, M.D.) . . . 143

SOCIETY: Decline in number of Fellows elected during 1895 lxxxvii

SPAIN: Parasite of malaria as observed in malarial fevers of south of Spain (Robert J. Marshall and George Thin, M.D.) 101

SPLEEN: Splenic anæmia (spléno-mégalie primitive) (Samuel West, M.D.) 323

STOMACH: see *Gastro-intestinal* and intestinal anastomosis.

SURGERY (operative): Posture in its relation to surgical operations under anæsthetics (Frederic Hewitt, M.D., and A. Marmaduke Sheild) 1

— — Neoplasm cyst of brain treated by operation (Charles A. Ballance) 167

SYPHILIS (infection): The exceptions to Colles' law (George Ogilvie, M.B.) 87

THIN, George, M.D.

A note on the appearances found in the tissues in a fatal case of pernicious malaria at Sierra Leone . 143

— and *MARSHALL, Robert J.*

The parasite of malaria as observed in the malarial fevers of the south of Spain 101

Communication of paper by Surg.-Capt. M. Louis Hughes on the endemic fever of the Mediterranean . 209

TREASURERS: Treasurers' Report . lxxxvi

TUMOUR: excision of portion of liver for tumour (Mayo Robson) 159

WEST, Samuel, M.D.

Case of splenic anæmia (spléno-mégalie primitive), with an account of the disease . . . 323

PRINTED BY ADLARD AND SON, BARTHOLOMEW CLOSE, E.C.